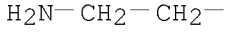
L1 ANSWER 140 OF 140 REGISTRY COPYRIGHT 2008 ACS on STN
RN 60-23-1 REGISTRY
ED Entered STN: 16 Nov 1984
CN Ethanethiol, 2-amino- (8CI, 9CI) (CA INDEX NAME)
OTHER NAMES:
CN β-Aminoethanethiol
CN β-Aminoethylthiol
CN β-MEA
CN β-Mercaptoethylamine
CN 1-Amino-2-mercaptoethane
CN 2-Amino-1-ethanethiol
CN 2-Aminoethanethiol
CN 2-Aminoethyl mercaptan
CN 2-Aminoethylthiol
CN 2-Mercaptoethanamine
CN 2-Mercaptoethylamine
CN Becaptan
CN Cysteamine
CN Cysteinamine
CN Decarboxycysteine
CN L 1573
CN Lambraten
CN Lambratene
CN MEA
CN MEA (mercaptan)
CN Mercamin
CN Mercamine
CN Mercaptamin
CN Mercaptamine
CN Mercaptoethylamine
CN Merkamin
CN NSC 647528
CN Riacon
CN Thioethanolamine
CN WR 347
DR 139720-70-0
MF C2 H7 N S
CI COM
LC STN Files: ADISINSIGHT, ADISNEWS, AGRICOLA, ANABSTR, AQUIRE, BEILSTEIN*, BIOSIS, BIOTECHNO, CA, CABA, CAOLD, CAPLUS, CASREACT, CBNB, CHEMCATS, CHEMINFORMRX, CHEMLIST, CIN, CSCHEM, CSNB, DDFU, DETHERM*, DRUGU, EMBASE, GMELIN*, HSDB*, IFICDB, IFIPAT, IFIUDB, IPA, MEDLINE, MRCK*, MSDS-OHS, PIRA, PROMT, PS, RTECS*, SYNTHLINE, TOXCENTER, USAN, USPAT2, USPATFULL, USPATOLD, VETU
(*File contains numerically searchable property data)
Other Sources: EINECS**, WHO
(**Enter CHEMLIST File for up-to-date regulatory information)



PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

6253 REFERENCES IN FILE CA (1907 TO DATE)
492 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
6263 REFERENCES IN FILE CAPLUS (1907 TO DATE)
75 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

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FULL ESTIMATED COST                                      29.11      29.32

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        608999 STRESS
        104143 STRESSES
        650123 STRESS
                  (STRESS OR STRESSES)
L2         123 L1 AND STRESS

=> s l2 and pd <=2004
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                  (PD<=20049999)
L3          88 L2 AND PD <=2004

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PROCESSING COMPLETED FOR L3
L4           88 FOCUS L3 1-

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L4   ANSWER 1 OF 88  CAPLUS  COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER:        1993:122175  CAPLUS
DOCUMENT NUMBER:         118:122175
ORIGINAL REFERENCE NO.:  118:21137a,21140a
TITLE:                   Stress and cysteamine-induced duodenal ulcer
AUTHOR(S):               Pare, W. P.; Bakke, H. K.; Kluczynski, J. M.
CORPORATE SOURCE:        Pavlovian Res. Lab., VA Med. Cent., Perry Point, MD,
                         USA
SOURCE:                  Experimental and Clinical Gastroenterology (
```

1991), 1(4), 299-306
CODEN: ECGAEQ; ISSN: 0353-9245
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Wistar Kyoto (WKY) normotenisve female rats received oral cysteamine HCl either at 20 mg/100 g or 30 mg/100 g and were subjected, 24 h later, to water-restraint (WR) stress for 2 h followed by a 2 h rest period. The incidence of restraint gastric stress ulcer was contingent on stress exposure and was independent of cysteamine. Duodenal ulceration required oral cysteamine, but ulcer severity was attenuated by stress. A chronic duodenal ulcer regimen (20 mg/100 g orally plus a 0.05% cysteamine drinking solution) plus exposure to daily uncontrollable tail shock failed to demonstrate the inhibitory effect of stress on cysteamine ulcer. In a final study, stress exposure, either before (in the form of foot shock) or after (in the form of WR) oral cysteamine HCL, 30 mg/100 g was effective in reducing duodenal ulcer severity as compared to non-stressed rats. Stress influences, not only restraint stress ulcer in the stomach, but also cysteamine-induced duodenal ulcer.
IT 60-23-1, Cysteamine
RL: BIOL (Biological study)
(duodenal ulcer induced by, stress effect on)
RN 60-23-1 CAPLUS
CN Ethanethiol, 2-amino- (8CI, 9CI) (CA INDEX NAME)

L4 ANSWER 2 OF 88 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2000:870541 CAPLUS
DOCUMENT NUMBER: 134:91616
TITLE: Changes in surface stress at the liquid/solid interface measured with a microcantilever
AUTHOR(S): Raiteri, R.; Butt, H.-J.; Grattarola, M.
CORPORATE SOURCE: Institute for Physical Chemistry, University of Mainz, Mainz, 55128, Germany
SOURCE: Electrochimica Acta (2000), 46(2-3), 157-163
CODEN: ELCAAV; ISSN: 0013-4686
PUBLISHER: Elsevier Science Ltd.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The bending of microfabricated silicon nitride cantilevers was used to determine surface stress changes at solid-liquid interfaces. The radius of curvature of the bent cantilever is directly proportional to changes in the differential surface stress between its opposite sides. To demonstrate the possibilities and limitations of the technique, cantilevers coated on both sides with Au and densely packed monolayers of different thiols were put in a constant flow of aqueous electrolyte solution and the deflection was measured using a optical lever technique. Changes in the surface stress for the different thiol monolayers due to specific proton adsorption are presented. Possible applications and improvements of this technique are discussed.
IT 60-23-1, 2-Aminoethanethiol
RL: DEV (Device component use); USES (Uses)
(measurement of surface stress at liquid/solid interface by microcantilevers coated by monolayers of)
RN 60-23-1 CAPLUS
CN Ethanethiol, 2-amino- (8CI, 9CI) (CA INDEX NAME)

$H_2N-CH_2-CH_2-SH$

REFERENCE COUNT: 57 THERE ARE 57 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 3 OF 88 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2002:465808 CAPLUS
DOCUMENT NUMBER: 137:24393
TITLE: Agents for ameliorating carbonyl stress
INVENTOR(S): Miyata, Toshio
PATENT ASSIGNEE(S): Kurokawa, Kiyoshi, Japan
SOURCE: PCT Int. Appl., 33 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002047677	A1	20020620	WO 2001-JP10891	20011212 <--
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
AU 2002022613	A5	20020624	AU 2002-22613	20011212 <--
PRIORITY APPLN. INFO.:			JP 2000-378112 A	20001212
			WO 2001-JP10891 W	20011212

AB Disclosed are agents for ameliorating carbonyl stress which comprise cysteamine or salts thereof. These agents are usable as drugs directly acting on carbonyl stress by bringing into contact with blood or a dialyzate during hemodialysis or peritoneal dialysis. These agents, which can be administered via the oral route etc., are also usable as drugs directly acting on carbonyl stress in vivo.

IT 60-23-1, Cysteamine
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(cysteamine for ameliorating carbonyl stress)

RN 60-23-1 CAPLUS
CN Ethanethiol, 2-amino- (8CI, 9CI) (CA INDEX NAME)

$H_2N-CH_2-CH_2-SH$

REFERENCE COUNT: 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 4 OF 88 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1997:462977 CAPLUS
DOCUMENT NUMBER: 127:145387
ORIGINAL REFERENCE NO.: 127:27941a,27944a
TITLE: Neurotrophic factors, cytokines and stress increase expression of basic fibroblast growth factor

in retinal pigmented epithelial cells
AUTHOR(S): Hackett, Sean F.; Schoenfeld, Carl-Ludwig; Freund, John; Gottsch, John D.; Bhargave, Sudeepta; Campochiaro, Peter A.
CORPORATE SOURCE: The Wilmer Eye Institute and the Department of Neuroscience, The Johns Hopkins University School of Medicine, Baltimore, MD, 21287-9277, USA
SOURCE: Experimental Eye Research (1997), 64(6), 865-873
CODEN: EXERA6; ISSN: 0014-4835
PUBLISHER: Academic
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Basic fibroblast growth factor (bFGF) and FGF receptors have been localized to photoreceptors and retinal pigmented epithelium (RPE), but the function of bFGF in adult retina and RPE is unknown. Exogenous bFGF has a neuroprotective effect in retina and brain and its expression is increased in some neurons in response to cytokines or stress. In this study, the authors investigated the effect of light, other types of stress, neurotrophic factors, and cytokines on bFGF levels in cultured human RPE. Some agents that protect photoreceptors from the damaging effects of constant light, including brain derived neurotrophic factor (BDNF), ciliary neurotrophic factor, and interleukin-1β, increase bFGF mRNA levels in RPE cells. Intense light and exposure to oxidizing agents also increase bFGF mRNA levels in RPE cells and cycloheximide blocks the increase. An increase in bFGF protein levels was demonstrated by ELISA in RPE cell supernatants after incubation with BDNF or exposure to intense light or oxidizing agents. These data indicate that bFGF is modulated in RPE cells by stress and by agents that provide protection from stress and support the hypothesis that bFGF functions as a survival factor in the outer retina.
IT 60-23-1, Cysteamine
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(neurotrophic factors, cytokines and stress increase expression of basic fibroblast growth factor in retinal pigmented epithelial cells)
RN 60-23-1 CAPLUS
CN Ethanethiol, 2-amino- (8CI, 9CI) (CA INDEX NAME)

$H_2N-CH_2-CH_2-SH$

REFERENCE COUNT: 54 THERE ARE 54 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 5 OF 88 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2002:133959 CAPLUS
DOCUMENT NUMBER: 137:163762
TITLE: Identification of α-dicarbonyl scavengers for cellular protection against carbonyl stress
AUTHOR(S): Wondrak, Georg T.; Cervantes-Laurean, Daniel; Roberts, Michael J.; Qasem, Jaber G.; Kim, Moonsun; Jacobson, Elaine L.; Jacobson, Myron K.
CORPORATE SOURCE: Arizona Cancer Center, College of Pharmacy, Department of Pharmacology and Toxicology, University of Arizona, Tucson, AZ, 85724, USA
SOURCE: Biochemical Pharmacology (2002), 63(3), 361-373
CODEN: BCPCA6; ISSN: 0006-2952

PUBLISHER: Elsevier Science Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Tissue deterioration and aging have long been associated with the accumulation of chemical induced protein and DNA damage. Reactive oxygen species (ROS) and reactive carbonyl species (RCS), especially α-dicarbonyl compds., are key mediators of damage caused by oxidative stress, glycation, and UV-irradiation The toxic effects of ROS are counteracted in vivo by antioxidants and antioxidant enzymes, and the deleterious effects of one RCS, methylglyoxal, are counteracted by a ubiquitous glyoxalase system. Carbonyl stress as a result of toxic effects of various mono-dicarbonyls (e.g. 4-hydroxynonenal) and α-dicarbonyls (e.g. glyoxal and deoxyosones) cannot be directly antagonized by antioxidants, and only a small number of biol. carbonyl scavengers like glutathione (GSH) have been identified to date. We have developed a new screening method for the identification of carbonyl scavengers using a rapid glycation system that proceeds independent of oxygen and therefore, excludes identification of inhibitory compds. acting as antioxidants. Using this screening assay adapted to 96-well microtiter plates, we have identified the cysteine derivative 3,3-dimethyl-D-cysteine as a potent inhibitor of non-oxidative advanced glycation. Comparative kinetic analyses demonstrated the superior α-oxoaldehyde-scavenging activity of D-penicillamine over that of aminoguanidine. D-Penicillamine traps α-oxoaldehydes by forming a 2-acylthiazolidine derivative as shown by structure elucidation of reaction products between D-penicillamine and methylglyoxal or phenylglyoxal. We demonstrated that upon co-incubation, D-penicillamine protects human skin keratinocytes and fibroblasts (CF3 cells) against glyoxal- and methylglyoxal-induced carbonyl toxicity. Our research qualifies α-amino-β-mercapto-β,β-dimethylethane as a promising pharmacophore for the development of related α-dicarbonyl scavengers as therapeutic agents to protect cells against carbonyl stress.
IT 60-23-1, Cysteamine
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(identification of α-dicarbonyl scavengers for cellular protection against carbonyl stress)
RN 60-23-1 CAPLUS
CN Ethanethiol, 2-amino- (8CI, 9CI) (CA INDEX NAME)

$H_2N-CH_2-CH_2-SH$

REFERENCE COUNT: 50 THERE ARE 50 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

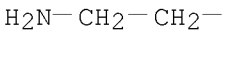
L4 ANSWER 6 OF 88 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1972:560385 CAPLUS
DOCUMENT NUMBER: 77:160385
ORIGINAL REFERENCE NO.: 77:26311a,26314a
TITLE: Inhibitory effect of cysteamine on the corticosterone content of the rat adrenal gland after stress
AUTHOR(S): Flemming, K.; Geierhaas, B.
CORPORATE SOURCE: Inst. Biophys. Strahlenbiol., Univ. Freiburg/Br., Freiburg, Fed. Rep. Ger.
SOURCE: Experientia (1972), 28(8), 965-6
CODEN: EXPEAM; ISSN: 0014-4754
DOCUMENT TYPE: Journal
LANGUAGE: German
AB The increase in the corticosterone [50-22-6] content of rat adrenal gland,

as induced by stress (irradiation), was inhibited by cysteamine-HCl (I) [156-57-0] (5 I.U./kg, i.p.). The inhibition of the corticosterone increase was not attributed to the compound's radioprotective effect since this inhibition was observed with I injection prior to or after irradiation I also inhibited the corticosterone increase induced by Na salicylate [54-21-7], histamine [51-45-6], or exogenous ACTH [9002-60-2]. Cystamine [51-85-4], but not cysteine [52-90-4], produced a similar effect as I in rats after stress.

IT 156-57-0
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(corticosterone of adrenal gland response to stress inhibition by)

RN 156-57-0 CAPLUS

CN Ethanethiol, 2-amino-, hydrochloride (1:1) (CA INDEX NAME)



L4 ANSWER 7 OF 88 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2004:999662 CAPLUS
DOCUMENT NUMBER: 141:406156
TITLE: Methods for reducing oxidative stress in a cell with a sulfhydryl protected glutathione prodrug
INVENTOR(S): Nagasawa, Herbert T.; Cohen, Jonathan F.
PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 12 pp.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 20040229815	A1	20041118	US 2003-750005	20031230 <--
AU 2004315267	A1	20050818	AU 2004-315267	20041227
CA 2552285	A1	20050818	CA 2004-2552285	20041227
WO 2005074903	A2	20050818	WO 2004-US43660	20041227
WO 2005074903	A3	20060223		
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RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
EP 1701732	A2	20060920	EP 2004-821314	20041227
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, PL, SK, BA, HR, IS, YU				

CN 1921876	A	20070228	CN 2004-80042221		20041227
IN 2006MN00915	A	20070330	IN 2006-MN915		20060731
PRIORITY APPLN. INFO.:			US 2003-437872P	P	20030103
			US 2003-750005	A	20031230
			WO 2004-US43660	W	20041227

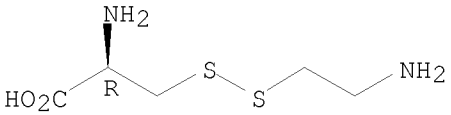
AB The invention relates to compns. and methods for reducing oxidative stress in a cell. The invention is comprised of contacting a cell with a sulfhydryl protected glutathione or cysteine prodrug thereby increasing intracellular glutathione or L-cysteine levels resulting in reduced hepatotoxicity.

IT 22801-37-2
RL: PKT (Pharmacokinetics); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(sulfhydryl protected glutathione prodrug reduces oxidative stress in cells)

RN 22801-37-2 CAPLUS

CN L-Alanine, 3-[(2-aminoethyl)dithio]- (9CI) (CA INDEX NAME)

Absolute stereochemistry.



L4 ANSWER 8 OF 88 CAPLUS COPYRIGHT 2008 ACS on STN

ACCESSION NUMBER:	1995:446171 CAPLUS
DOCUMENT NUMBER:	122:236066
ORIGINAL REFERENCE NO.:	122:43047a,43050a
TITLE:	A cellular stress model for the sequestration of redox-active glial iron in the aging and degenerating nervous system
AUTHOR(S):	Wang, Xudong; Manganaro, Fortunato; Schipper, Hyman M.
CORPORATE SOURCE:	Bloomfield Cent. Res. Aging, Lady Davis Inst. Med. Res., Montreal, QC, Can.
SOURCE:	Journal of Neurochemistry (1995), 64(4), 1868-77 CODEN: JONRA9; ISSN: 0022-3042
PUBLISHER:	Lippincott-Raven
DOCUMENT TYPE:	Journal
LANGUAGE:	English

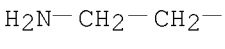
AB The mechanisms responsible for the accumulation of redox-active brain iron in normal senescence and in Parkinson's disease remain poorly understood. The aminothiol compound cysteamine (CSH) induces the appearance of autofluorescent, iron-rich cytoplasmic granules in cultured astroglia that are identical to glial inclusions that progressively accumulate in the aging periventricular brain. Both in situ and in culture, these glial inclusions appear to arise in the context of a generalized cellular stress (heat shock) response. Several labs. have previously concluded that porphyrins and heme ferrous iron are responsible, resp., for red-orange autofluorescence and nonenzymic peroxidase activity in the glial inclusions. In the present study we found that, contrary to hypothesis, CSH suppresses the incorporation of the heme precursors δ-amino[14C]levulinic acid and [14C]glycine into astroglial porphyrin and heme in primary culture. Similar results were obtained when the cells were preloaded with radiolabeled heme precursors for 24 h before CSH treatment, suggesting that the latter directly inhibits porphyrin-heme biosynthesis rather than limiting precursor uptake by these cells. The authors also demonstrated that CSH exposure results in the sequestration

of iron-59 by astroglial mitochondria (granule precursors). The results of this study suggest that stress-related trapping of nonheme iron by astroglial mitochondria may be an important mechanism underlying the pathol. accumulation of redox-active iron in the basal ganglia of subjects with Parkinson's disease. CSH-treated astrocytes provide a useful model investigate the role of stress-related dysregulation of neuroglial iron metabolism in the aging and degenerating nervous system.

IT 60-23-1, Cysteamine
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(iron-rich cytoplasmic granules accumulation in astrocytes response to cysteamine in cellular stress model)

RN 60-23-1 CAPLUS

CN Ethanethiol, 2-amino- (8CI, 9CI) (CA INDEX NAME)

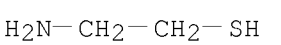


L4 ANSWER 9 OF 88 CAPLUS COPYRIGHT 2008 ACS on STN

ACCESSION NUMBER: 1994:28071 CAPLUS
DOCUMENT NUMBER: 120:28071
ORIGINAL REFERENCE NO.: 120:5221a,5224a
TITLE: Stress protein co-localization to autofluorescent astrocytic inclusions in situ and in cysteamine-treated glial cultures
AUTHOR(S): Mydlarski, Marc B.; Schipper, Hyman M.
CORPORATE SOURCE: Bloomfield Centre for Research in Aging, Lady Davis Institute for Medical Research, Sir Mortimer B. Davis-Jewish General Hospital, Department of Neurology and Neurosurgery, and Centre for Studies in Aging, McGill University, Montreal, Que., Can.
SOURCE: Brain Research (1993), 627(1), 113-21
CODEN: BRREAP; ISSN: 0006-8993
DOCUMENT TYPE: Journal
LANGUAGE: English

AB In the aging brain, a unique subpopulation of limbic and periventricular astrocytes accumulates red autofluorescent, peroxidase-pos. cytoplasmic inclusions distinct from lipofuscin. Cysteamine (CSH) exposure rapidly induces identical inclusions in cultured, immature astroglia. CSH induces a cellular stress response prior to astrocyte granulation. To determine whether stress proteins are actual constituents of the autofluorescent granules, 12-wk-old rat brain sections and CSH-treated astroglial cultures were immunostained with various anti-stress protein antibodies and evaluated by laser scanning confocal microscopy. The authors observed intense co-localization of heat shock protein (HSP) 27 and ubiquitin (Ub) to the autofluorescent astrocyte granules in situ and in CSH-treated glial cultures. In both prepns., glucose-regulated protein (GRP) 94 consistently exhibited partial co-localization to the granule periphery and adjacent cytoplasm. In contrast, HSP72 co-localization to these inclusions was only occasionally seen and the granules appeared entirely devoid of HSP90 and αB-crystallin. Acute exposure of cultured astroglia to CSH induced intense cytoplasmic Ub staining, suggesting that activation of the Ub pathway may be an early event in the biogenesis of these astrocytic granules. Taken together, the authors' results support the notion that the autofluorescent astrocyte inclusions are stress or heat shock granules which progressively accumulate in the aging periventricular brain. Moreover, CSH greatly accelerates the appearance of this senescent astrocyte phenotype in primary culture.

IT 60-23-1, Cysteamine
RL: BIOL (Biological study)
(autofluorescent granule induced by, in immature astroglia in primary culture, stress proteins in relation to)
RN 60-23-1 CAPLUS
CN Ethanethiol, 2-amino- (8CI, 9CI) (CA INDEX NAME)



L4 ANSWER 10 OF 88 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1994:28644 CAPLUS
DOCUMENT NUMBER: 120:28644
ORIGINAL REFERENCE NO.: 120:5365a
TITLE: Role of the cellular stress response in the biogenesis of cysteamine-induced astrocytic inclusions in primary culture
AUTHOR(S): Mydlarski, Marc B.; Liang, Jin Jun; Schipper, Hyman M.
CORPORATE SOURCE: Cent. Stud. Aging, McGill Univ., Montreal, QC, Can.
SOURCE: Journal of Neurochemistry (1993), 61(5), 1755-65
CODEN: JONRA9; ISSN: 0022-3042
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Cysteamine (CSH; 2-mercaptoethylamine) stimulates the accumulation of peroxidase-pos. inclusions in cultured astroglia akin to those observed in the aging periventricular brain. Because CSH induces the synthesis of a stress protein (heme oxygenase) in rat liver, the authors hypothesized that aspects of the cellular stress response may play a role in the biogenesis of CSH-induced astrocyte granules. In the present study, the authors performed indirect immunofluorescent staining and immunoblotting for various stress proteins in rat neuroglial cultures. Exposure of astrocyte cultures to CSH enhanced immunostaining for heme oxygenase-1 (HO-1) and heat-shock proteins 27, 72, and 90, but not glucose-regulated protein 94, relative to untreated cultures. CSH-pretreated astrocytes exhibited enhanced tolerance to H2O2 toxicity relative to untreated cells, providing physiol. evidence of an antecedent stress response in the former. In addition, exposure for 12 days to H2O2, a known inducer of the stress response, elicited astrocyte granulation similar to that observed with CSH. Chronic induction of HO-1 and other stress proteins may participate in the biogenesis of metalloporphyrin-rich inclusions in CSH-treated astroglial cultures and in astrocytes of the aging periventricular brain.
IT 60-23-1, Cysteamine
RL: BIOL (Biological study)
(stress protein formation response. to, in astrocytes, metalloporphyrin-rich inclusions in relation to)
RN 60-23-1 CAPLUS
CN Ethanethiol, 2-amino- (8CI, 9CI) (CA INDEX NAME)

L4 ANSWER 11 OF 88 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2007:1090326 CAPLUS
DOCUMENT NUMBER: 147:336379
TITLE: Therapeutic and prophylactic uses of cell specific

carbonic anhydrase enzymes in treating aging disorders due to oxidative stress and as growth factors of stem cells

INVENTOR(S): Rodriguez, Victorio C.

PATENT ASSIGNEE(S): USA

SOURCE: U.S. Pat. Appl. Publ., 13pp., Cont.-in-part of U.S. Ser. No. 858,091.
CODEN: USXXCO

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 4

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 20070224182	A1	20070927	US 2007-801870	20070512
US 6821997	B1	20041123	US 2002-77719	20020215 <--
US 20040253223	A1	20041216	US 2004-858091	20040601 <--
US 7256184	B2	20070814		

PRIORITY APPLN. INFO.:

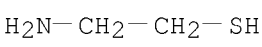
US 2000-688290	B2	20001016
US 2002-77719	A2	20020215
US 2004-858091	A2	20040601

AB A method for the treatment and prophylaxis of conditions of aging due oxidative stress and as growth factors of stem cells. Such conditions due to oxidative stress are associated with a decreased presence of one or more cell-specific carbonic anhydrase enzymes in the tissue of a subject. Such conditions include but are not limited to Alzheimer's disease, Parkinson's disease, multiple sclerosis, autism, Lou Gehrig's disease, Huntington's disease, diabetes mellitus, amyloid diseases, atherosclerosis, arthritis, osteoporosis, cystic fibrosis. The method comprises administering to the patient a pharmaceutically effective, non-toxic amount of one or more compds. that increases the presence of one or more Carbonic Anhydrase Isoenzymes whose levels have been reduced in the subject. Such compound maybe the Cell Specific Carbonic Anhydrase Enzymes, a compound that when absorbed reacts or dissocs. to form cell specific carbonic enzymes or a compound that when administered promotes the natural generation of the cell specific carbonic anhydrase enzymes within the subject. This method also uses one or more cells specific carbonic anhydrase as growth factors of stem cells for replacing tissues due to injuries or diseases in humans. These methods includes the administering of these compds. over an extended period of time ranging from 6 mo until the subject dies.

IT 60-23-1, Cysteamine
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(therapeutic and prophylactic uses of cell specific carbonic anhydrase enzymes in treating aging disorders due to oxidative stress and as growth factors of stem cells)

RN 60-23-1 CAPLUS

CN Ethanethiol, 2-amino- (8CI, 9CI) (CA INDEX NAME)



L4 ANSWER 12 OF 88 CAPLUS COPYRIGHT 2008 ACS on STN

ACCESSION NUMBER: 1997:723130 CAPLUS

DOCUMENT NUMBER: 128:2307

ORIGINAL REFERENCE NO.: 128:511a,514a

TITLE: A cellular stress model for the differential

expression of glial lysosomal cathepsins in the aging nervous system

AUTHOR(S): Chopra, Vikrajmit S.; Moozar, Kouros L.; Mehindate, Khalil; Schipper, Hyman M.

CORPORATE SOURCE: Bloomfield Centre for Research in Aging, Lady Davis Institute for Medical Research, Sir Mortimer B. Davis-Jewish General Hospital, McGill University, Montreal, QC, H3T 1E2, Can.

SOURCE: Experimental Neurology (1997), 147(2), 221-228
CODEN: EXNEAC; ISSN: 0014-4886

PUBLISHER: Academic

DOCUMENT TYPE: Journal

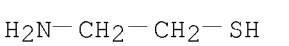
LANGUAGE: English

AB Activation of the endosomal-lysosomal system and altered expression of various lysosomal hydrolases have been implicated in several senescence-dependent neurodegenerative disorders and occurs, to a lesser extent, in the course of normal brain aging. The progressive accumulation of autofluorescent, peroxidase-pos. astrocytic granules represents a highly consistent biomarker of aging in the vertebrate CNS. The sulfhydryl agent cysteamine greatly accelerates the accumulation of these glial inclusions in situ and in primary brain cell cultures. We previously determined that these glial inclusions are derived from abnormal mitochondria which undergo fusion with lysosomal elements in a complex autophagic process. In the present study, we demonstrate that cysteamine suppresses cathepsin B mRNA levels and immunoreactive protein in cultured astroglia, whereas cathepsin D mRNA and protein levels are significantly augmented by CSH exposure in these cells. Moreover, cathepsin D (but not cathepsin B) exhibits robust colocalization to the red autofluorescent inclusions. Concordant with our in vitro observations, cathepsin B immunoreactivity is prominent in the hypothalamic ventromedial nucleus which accumulates few autofluorescent glial inclusions during aging and is relatively inapparent in the heavily granulated hypothalamic arcuate nucleus. Conversely, cathepsin D is prominent in the aging arcuate nucleus where it colocalizes to the autofluorescent inclusions and exhibits scant immunoreactivity in the adjacent ventromedial nuclear complex. In senescent astroglia, oxidative stress may down-regulate the cathepsin B gene as part of a concerted cellular stress (heat shock) response. Glial cathepsin D, on the other hand, resists stress-related inhibition and may play an important role in disposing of oxidatively modified mitochondria in the aging and degenerating nervous system.

IT 60-23-1, Cysteamine
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(cellular stress model for differential expression of glial lysosomal cathepsins in aging nervous system)

RN 60-23-1 CAPLUS

CN Ethanethiol, 2-amino- (8CI, 9CI) (CA INDEX NAME)



REFERENCE COUNT: 54 THERE ARE 54 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 13 OF 88 CAPLUS COPYRIGHT 2008 ACS on STN

ACCESSION NUMBER: 2004:637060 CAPLUS

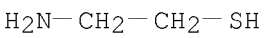
DOCUMENT NUMBER: 141:137690

TITLE: Vanin-1-/- mice exhibit a glutathione-mediated tissue

resistance to oxidative stress
AUTHOR(S): Berruyer, C.; Martin, F. M.; Castellano, R.; Macone, A.; Malergue, F.; Garrido-Urbani, S.; Millet, V.; Imbert, J.; Dupre, S.; Pitari, G.; Naquet, P.; Galland, F.
CORPORATE SOURCE: Centre d'Immunologie de Marseille-Luminy CNRS-INSERM-Universite de la Mediterranee, Marseille, 13288, Fr.
SOURCE: Molecular and Cellular Biology (2004), 24(16), 7214-7224
CODEN: MCEBD4; ISSN: 0270-7306
PUBLISHER: American Society for Microbiology
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Vanin-1 is an epithelial ectoenzyme with pantetheinase activity and generating the amino-thiol cysteamine through the metabolism of pantothenic acid (vitamin B5). Here we show that Vanin-1-/- mice, which lack cysteamine in tissues, exhibit resistance to oxidative injury induced by whole-body γ-irradiation or paraquat. This protection is correlated with reduced apoptosis and inflammation and is reversed by treating mutant animals with cysteamine. The better tolerance of the Vanin-1-/- mice is associated with an enhanced gamma-glutamylcysteine synthetase activity in liver, probably due to the absence of cysteamine and leading to elevated stores of glutathione (GSH), the most potent cellular antioxidant. Consequently, Vanin-1-/- mice maintain a more reducing environment in tissue after exposure to irradiation In normal mice, we found a stress-induced biphasic expression of Vanin-1 regulated via antioxidant response elements in its promoter region. This process should finely tune the redox environment and thus change an early inflammatory process into a late tissue repair process. We propose Vanin-1 as a key mol. to regulate the GSH-dependent response to oxidative injury in tissue at the epithelial level. Therefore, Vanin/pantetheinase inhibitors could be useful for treatment of damage due to irradiation and pro-oxidant inducers.

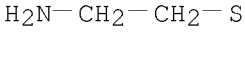
IT 60-23-1, Cysteamine
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(vanin-1-/- mice, which lack cysteamine in tissues, exhibit glutathione-mediated tissue resistance to oxidative stress evoked by irradiation and pro-oxidant inducers and reduction of apoptosis and inflammation)
RN 60-23-1 CAPLUS
CN Ethanethiol, 2-amino- (8CI, 9CI) (CA INDEX NAME)



REFERENCE COUNT: 41 THERE ARE 41 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 14 OF 88 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1995:801378 CAPLUS
DOCUMENT NUMBER: 123:234440
ORIGINAL REFERENCE NO.: 123:41755a,41758a
TITLE: Effect of minor elements on intergranular stress-corrosion cracking of carbon steel in aqueous amine solution
AUTHOR(S): Togashi, Kiyohide; Sugimoto, Katsuhisa
CORPORATE SOURCE: Idemitsu Eng. Co., Ltd., Chiba, 260, Japan
SOURCE: Zairyo to Kankyo (1995), 44(8), 430-5
CODEN: ZAKAEP; ISSN: 0917-0480

PUBLISHER: Japan Society of Corrosion Engineering
DOCUMENT TYPE: Journal
LANGUAGE: Japanese
AB The effect of minor elements on the intergranular stress -corrosion cracking (IGSCC) of carbon steel in an aqueous amine solution has been studied using slow strain rate testing (SSRT) technique at potentials in an active-passive transition range. A series of carbon steels with varying content of minor elements such as C, N, P, S and Si were used as specimens. The SSRT was carried out at a strain rate of 1.11 + 10-6/s in a 20 mass% MEA solution of pH 8 at 333 K. The IGSCC of the carbon steels depended upon potentials and susceptibility maxima to the IGSCC were attained at potentials in the center of active-passive transition ranges. The susceptibility to the IGSCC increased with decreasing C and N content and with increasing P and Si content of the carbon steels. The susceptibility to the IGSCC hardly changed with increasing S content of the carbon steels.
IT 60-23-1, MEA
RL: NUU (Other use, unclassified); USES (Uses)
(effect of minor elements on intergranular stress-corrosion cracking in aqueous amine)
RN 60-23-1 CAPLUS
CN Ethanethiol, 2-amino- (8CI, 9CI) (CA INDEX NAME)

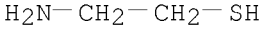


L4 ANSWER 15 OF 88 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2000:711933 CAPLUS
DOCUMENT NUMBER: 134:350036
TITLE: 2-Mercaptoethylamine, radioprotector, inhibits the induction of the oxidative stress-inducible (soi) gene by paraquat in Escherichia coli
AUTHOR(S): Gyu Kim, In; Jeong Oh, Tae
CORPORATE SOURCE: Department of Radiation Biology, Environmental Radiation Research Group, Korea Atomic Energy Research Institute, Yusong, Taejon, 305-600, S. Korea
SOURCE: Pharmacological Research (2000), 42(5), 429-433
CODEN: PHMREP; ISSN: 1043-6618
PUBLISHER: Academic Press
DOCUMENT TYPE: Journal
LANGUAGE: English
AB To demonstrate the ·O2--scavenging activity of 2-mercaptoethylamine (MEA), the induction of the oxidative stress-inducible (soi) gene-fused lacZ gene (soi-28::lacZ) was investigated by the use of paraquat as a source of ·O2-. When MEA or cysteine was added to E. coli cultures before paraquat treatment, soi gene induction by paraquat was inhibited. A high concentration of ascorbic acid (5 mm) inhibited soi gene induction by paraquat far less than than did MEA or cysteine. The inhibition of soi gene induction by MEA was concentration dependent. Mols. which antagonize the radioprotective action of MEA, ascorbic acid and cysteine, did not counteract the effect of MEA on the inhibition of paraquat-mediated soi gene induction. To clarify that the action of MEA on the inhibition of paraquat-mediated soi gene induction may be due, in part, to ·O2--scavenging activity, expts. investigated the ability of MEA to inhibit the nitroblue tetrazolium (NBT) reduction mediated by ·O2- generated in the xanthine oxidase/hypoxanthine system in

vitro. At concns. >1 mm, MEA effectively inhibited NBT reduction in a concentration-dependent fashion. The results demonstrated that MEA has an ability to scavenge ·O2-, and so it protects against ·O2--mediated damage. (c) 2000 The Italian Pharmacological Society.

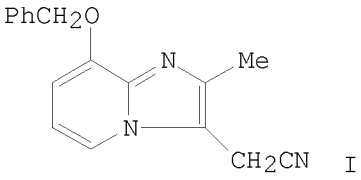
IT 60-23-1, 2-Mercaptoethylamine
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(mrcaptoethylamine inhibition of the induction of the oxidative stress-inducible (soi) gene by paraquat in Escherichia coli)

RN 60-23-1 CAPLUS
CN Ethanethiol, 2-amino- (8CI, 9CI) (CA INDEX NAME)



REFERENCE COUNT: 28 THERE ARE 28 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 16 OF 88 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1984:583790 CAPLUS
DOCUMENT NUMBER: 101:183790
ORIGINAL REFERENCE NO.: 101:27653a,27656a
TITLE: Effects of a gastric antisecretory-cytoprotectant 2-methyl-8-(phenylmethoxy)imidazo[1,2-a]pyridine-3-acetonitrile (Sch 28 080) on cysteamine, reserpine and stress ulcers in rats
AUTHOR(S): Chiu, P. J. S.; Gerhart, C.; Brown, A. D.; Barnett, A.
CORPORATE SOURCE: Dep. Pharmacol., Schering-Plough Corp., Bloomfield, NJ, 07003, USA
SOURCE: Arzneimittel-Forschung (1984), 34(7), 783-6
CODEN: ARZNAD; ISSN: 0004-4172
DOCUMENT TYPE: Journal
LANGUAGE: English
GI



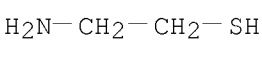
AB PGE2 [363-24-6] and carbenoxolone [5697-56-3], putative cytoprotective agents, were tested in cysteamine [60-23-1], reserpine [50-55-5] and stress ulcers in rats. In cysteamine-induced duodenal ulcer, PGE2 was inactive at 0.1 and 0.5 mg/kg, orally; carbenoxolone at 100 mg/kg, orally, decreased the incidence but not the severity of the ulcer. PGE2 at 5.0 mg/kg, orally, and carbenoxolone at 300 mg/kg, orally, showed moderate effects, but the dosage also inhibited cysteamine-stimulated acid secretion. PGE2 (0.1 and 0.3 mg/kg, orally) was inactive and carbenoxolone (100 and 300 mg/kg, orally) further aggravated the gastric ulceration caused by reserpine or cold-restraint stress. In contrast, atropine [51-55-8] (3 and 10 mg/kg, orally) and cimetidine [51481-61-9] (30, 100, and 300 mg/kg, orally) were active

in all 3 ulcer models. But the results with cimetidine in stress ulcer were somewhat variable. Sch 28080 (2-methyl-8-(phenylmethoxy)imidazo[1,2-a]pyridine-3-acetonitrile)(I) [76081-98-6], a novel structure with both cytoprotective and antisecretory activity, was highly efficacious in cysteamine, reserpine, and stress ulcers (1-30 mg/kg, orally), which was presumably adequately accounted for by its potent antisecretory activity. Apparently, cysteamine, reserpine, and stress ulcers may not be appropriate models for testing the potential antiulcer effect of primarily cytoprotective compds.

IT 60-23-1
RL: BIOL (Biological study)
(ulcer from, gastric antisecretory-cytoprotective agents effect on, as animal model)

RN 60-23-1 CAPLUS

CN Ethanethiol, 2-amino- (8CI, 9CI) (CA INDEX NAME)



L4 ANSWER 17 OF 88 CAPLUS COPYRIGHT 2008 ACS on STN

ACCESSION NUMBER: 1990:77171 CAPLUS

DOCUMENT NUMBER: 112:77171

ORIGINAL REFERENCE NO.: 112:13195a,13198a

TITLE: Preparation and formulation of 2-thiazolidinone derivatives and their use for treatment of gastric and duodenal ulcers

INVENTOR(S): Szabadkai, Istvan; Harsanyi, Kalman; Lampert, Agnes; Domany, Gyorgy; Hegedus, Bela; Kapolnas Pap, Marta; Ezer, Elemer; Matuz, Judit; Saghy, Katalin; et al.

PATENT ASSIGNEE(S): Richter, Gedeon, Vegyeszeti Gyar Rt., Hung.

SOURCE: Eur. Pat. Appl., 14 pp.
CODEN: EPXXDW

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

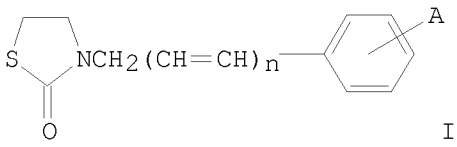
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
EP 320910	A1	19890621	EP 1988-120908	19881214 <--
EP 320910	B1	19931208		
R: AT, BE, CH, DE, ES, FR, GB, GR, IT, LI, NL, SE				
HU 49125	A2	19890828	HU 1987-5632	19871214 <--
HU 198915	B	19891228		
CA 1332419	C	19941011	CA 1988-585257	19881207 <--
CN 1033625	A	19890705	CN 1988-108626	19881213 <--
CN 1019196	B	19921125		
JP 02000279	A	19900105	JP 1988-314813	19881213 <--
US 4937252	A	19900626	US 1988-283809	19881213 <--
SU 1657062	A3	19910615	SU 1988-4613151	19881214 <--
AT 98235	T	19931215	AT 1988-120908	19881214 <--
ES 2049243	T3	19940416	ES 1988-120908	19881214 <--

PRIORITY APPLN. INFO.: HU 1987-5632 A 19871214
EP 1988-120908 A 19881214

OTHER SOURCE(S): MARPAT 112:77171

GI

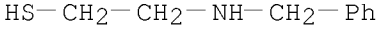


I

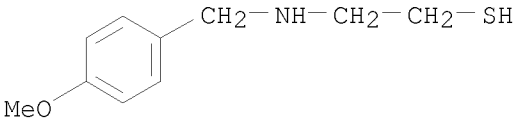
AB Title compds. I (A = H, halo, C1-4 alkyl, C1-4 alkoxy, NO2; n = 0, 1), are prepared I show a cytoprotective and gastric acid secretion-inhibiting effect and thus may be used in the therapy of gastric and duodenal ulcers. I may be prepared e.g., by reacting a cysteamine derivative with a carbonic acid derivative HSCH2CH2NHCH2Ph and (PhO)2CO in EtOH was refluxed under N for 24 h to give 56.4% I (A = H; n = 0) (II). II prevented gastric ulcer induced by indomethacin, aspirin, and aspirin + stress with ED50 of 1.0, 1.3, and 22.0 mg/kg orally, resp. A tablet formulation (1000 tablets) comprised II 50, lactose 200, starch 32, and Mg stearate 3 g.

IT 5978-34-7, N-(Phenylmethyl)cysteamine 91251-87-5, N-(4-Methoxyphenylmethyl)cysteamine 94776-94-0, N-(4-Methylphenylmethyl)cysteamine 124622-06-6, N-(2-Chlorophenylmethyl)cysteamine
RL: RCT (Reactant); RACT (Reactant or reagent)
(cyclocondensation of, with di-Ph carbonate)

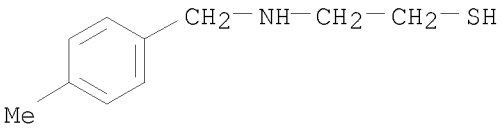
RN 5978-34-7 CAPLUS
CN Ethanethiol, 2-[(phenylmethyl)amino]- (CA INDEX NAME)



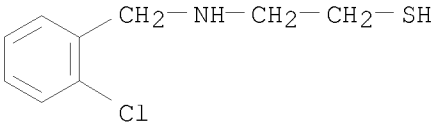
RN 91251-87-5 CAPLUS
CN Ethanethiol, 2-[[(4-methoxyphenyl)methyl]amino]- (CA INDEX NAME)



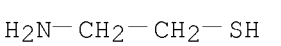
RN 94776-94-0 CAPLUS
CN Ethanethiol, 2-[[(4-methylphenyl)methyl]amino]- (CA INDEX NAME)



RN 124622-06-6 CAPLUS
CN Ethanethiol, 2-[[(2-chlorophenyl)methyl]amino]- (CA INDEX NAME)



L4 ANSWER 18 OF 88 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1992:233080 CAPLUS
DOCUMENT NUMBER: 116:233080
ORIGINAL REFERENCE NO.: 116:39435a,39438a
TITLE: Central dopamine involvement in experimental gastrointestinal injury
AUTHOR(S): Glavin, Gary B.
CORPORATE SOURCE: Fac. Med., Univ. Manitoba, Winnipeg, MB, R3E 0W3, Can.
SOURCE: Progress in Neuro-Psychopharmacology & Biological Psychiatry (1992), 16(2), 217-21
CODEN: PNPPD7; ISSN: 0278-5846
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Rats were prepared with intracerebral cannulas for microinjection of test compds. into various brain regions. Selective dopamine D1 agonists (SKF38393, SKF75670C) and a D1 antagonist (SCH23390) were injected into the cell body regions of the nigrostriatal, mesolimbic and mesocortical dopamine tracts or into a terminal field of these tracts (caudate nucleus, central nucleus of the amygdala and medial prefrontal cortex) prior to gastric ulcer induction by cold-restraint stress or duodenal ulcer induction by cysteamine. The dopamine D1 agonists reduced both stress gastric ulcers and duodenal lesions most significantly when given into either the cell body region or a terminal field of the mesolimbic DA tract with much less effects seen for the nigrostriatal tract. No effects were seen upon infusion of the agonists into the mesocortical cell body or terminal field regions. The D1 antagonist worsened both stress-induced gastric lesions and duodenal lesions if given into mesolimbic regions and, to a much lesser extent when injected into the nigrostriatal tract. No effect of the D1 antagonist was seen upon administration into the mesocortical tract. Central dopamine D1 receptors, particularly in the mesolimbic DA tract, appear to be involved in mediating the gastrointestinal consequences of exposure to stress.
IT 60-23-1, Cysteamine
RL: BIOL (Biological study)
(duodenal ulcer formation induced by, central dopaminergic D1 receptors of mesolimbic dopaminergic tract in)
RN 60-23-1 CAPLUS
CN Ethanethiol, 2-amino- (8CI, 9CI) (CA INDEX NAME)



L4 ANSWER 19 OF 88 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1986:620418 CAPLUS
DOCUMENT NUMBER: 105:220418
ORIGINAL REFERENCE NO.: 105:35470h,35471a
TITLE: Increase of glutathione biosynthesis after oxidative stress induced by thiols
AUTHOR(S): Issels, R.; Nagele, A.; Bourier, S.; Boening, B.; Wilmanns, W.
CORPORATE SOURCE: Inst. Haematol., GSF, Fed. Rep. Ger.
SOURCE: Superoxide Superoxide Dismutase Chem., Biol. Med., Proc. Int. Conf., 4th (1986), Meeting Date 1985, 419-21. Editor(s): Rotilio, Giuseppe. Elsevier: Amsterdam, Neth.
CODEN: 55GJAL
DOCUMENT TYPE: Conference

LANGUAGE: English

AB Cysteamine [60-23-1] (0.4 mM) exposure of CHO cells for 1 h at 37° resulted in a marked increase in 35S-labeled cystine [56-89-3] uptake from the medium into the cells. This increase paralleled a pronounced elevation in intracellular GSH [70-18-8] content. Both effects of cysteamine were completely blocked by incubation of cells at 5° during the time of drug treatment. Apparently, thiols like cysteamine promote cystine uptake in CHO cells followed by an increase in biosynthesis. The increased generation of activated O species during thiol autoxidn. which further react with elevated GSH levels could be an important step in the expression of the biol. effects of thiol-induced oxidative stress.

IT 60-23-1
RL: BIOL (Biological study)
(GSH formation response to, in CHO cells, cystine uptake in relation to)

RN 60-23-1 CAPLUS

CN Ethanethiol, 2-amino- (8CI, 9CI) (CA INDEX NAME)

L4 ANSWER 20 OF 88 CAPLUS COPYRIGHT 2008 ACS on STN

ACCESSION NUMBER: 2001:629807 CAPLUS

DOCUMENT NUMBER: 135:206701

TITLE: Mechanisms for the cytotoxicity of cysteamine

AUTHOR(S): Jeitner, Thomas M.; Lawrence, David A.

CORPORATE SOURCE: Wadsworth Center, New York State Department of Health, Albany, NY, 12201-0509, USA

SOURCE: Toxicological Sciences (2001), 63(1), 57-64
CODEN: TOSCF2; ISSN: 1096-6080

PUBLISHER: Oxford University Press

DOCUMENT TYPE: Journal

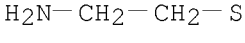
LANGUAGE: English

AB The major aim of this study was to quant. assess the contribution of H2O2 generation to the cytotoxicity induced by cysteamine. Cysteamine produces H2O2 at levels that correlate with its toxicity between 23 and 160 μM. A maximum of 6.9 μM H2O2 is generated by 625 μM cysteamine. When compared to the toxicity of exogenous H2O2, cysteamine-derived peroxide accounted for 57% of its toxicity. This corresponded to the percent toxicity due to 23 to 91 μM cysteamine. The remaining 43% toxicity appears to involve the inhibition of glutathione peroxidase, because activity of both the cellular and purified enzyme were inhibited by 200 μM cysteamine concns. CCRF-CEM cells have no catalase activity, so the inhibition of glutathione peroxidase may sensitize these cells to the less than toxic levels of peroxide generated by this aminothiol. Cysteamine also stimulated the production of cellular glutathione in a manner that was not related to its H2O2 generation. The production of glutathione did not influence toxicity but may reflect the accumulation of cysteamine to levels that inhibit glutathione peroxidase.

IT 60-23-1, Cysteamine
RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)
(cytotoxicity of cysteamine and mechanisms in relation to oxidative stress)

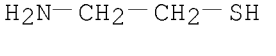
RN 60-23-1 CAPLUS

CN Ethanethiol, 2-amino- (8CI, 9CI) (CA INDEX NAME)



REFERENCE COUNT: 27 THERE ARE 27 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 21 OF 88 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1993:20311 CAPLUS
DOCUMENT NUMBER: 118:20311
ORIGINAL REFERENCE NO.: 118:3793a,3796a
TITLE: Functional and structural changes in the jejunum of the rat following cysteamine and stress -induced duodenal ulcer
AUTHOR(S): Maaluf, Vera Lucia M.; Atallah, Juliana B.; Nuwayri-Salti, Nuha; Abu Alfa, Amer K.; Nassar, Camille F.
CORPORATE SOURCE: Dep. Physiol., Am. Univ. Beirut, Beirut, Lebanon
SOURCE: Digestion (1992), 52(1), 13-19
CODEN: DIGEBW; ISSN: 0012-2823
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The effects of cysteamine and stress-induced duodenal ulcer on the functional and structural properties of the rat jejunum were investigated. The absorptive capacity of the jejunum was determined using alanine as the permeant solute and the single-pass perfusion technique. A decrease in alanine absorption was observed after 8 h and 3 days of duodenal ulcer induction by stress and cysteamine resp. However, alanine transport measured 7 days after cysteamine or stress ulcer induction showed no change from control values. Cysteamine and stress-induced duodenal ulcer did not show any change in water absorption across the jejunum when measured after 8 h, 3 and 7 days of ulcer induction. Microscopically, the jejunum of rats with 3-day cysteamine-induced ulcer exhibited diffuse type of apical derangements with excessive swelling of the villi and progressive degeneration changes. No such changes were noticed on the 7th day nor in the jejunum of the rats with stress-induced duodenal ulcer. Apparently, cysteamine-induced duodenal ulcer produces an inhibition in the absorptive capacity of the jejunum which is time-dependent and reversible.
IT 60-23-1, Cysteamine
RL: BIOL (Biological study)
(ulcer stimulation by, in duodenum, jejunum absorptive capacity response to)
RN 60-23-1 CAPLUS
CN Ethanethiol, 2-amino- (8CI, 9CI) (CA INDEX NAME)



L4 ANSWER 22 OF 88 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1987:437254 CAPLUS
DOCUMENT NUMBER: 107:37254
ORIGINAL REFERENCE NO.: 107:6203a,6206a
TITLE: Influence of oxidative stress induced by cysteamine upon the induction and development of thermotolerance in Chinese hamster ovary cells
AUTHOR(S): Issels, Rolf D.; Bourier, Susanne; Boening, Beatrice; Li, Gloria C.; Mak, John J.; Wilmanns, Wolfgang
CORPORATE SOURCE: Inst. Haematol., Ges. Strahlen- Umweltforsch., Munich, 8000, Fed. Rep. Ger.
SOURCE: Cancer Research (1987), 47(9), 2268-74
CODEN: CNREA8; ISSN: 0008-5472
DOCUMENT TYPE: Journal

LANGUAGE: English

AB Chinese hamster ovary cells exposed to the sulfhydryl compound cysteamine combined with heat treatment at 44° developed thermotolerance within 8 h. After initial treatment either with 15 min cysteamine (0.4 mM) at 37° immediately followed by 15 min heat at 44° or with 15 min cysteamine (0.4 mM) at 44°, the magnitude of thermotolerance developed was identical. The D0 (minutes of heat treatment which reduce the surviving fraction by factor e (67%) on the exponential portion of the survival curve) of the subsequent 44° heat survival curves increased by factors of 8.9 and 7.9, resp. The kinetics of thermotolerance induction and the time to reach the maximum of thermotolerance expression after combined cysteamine treatment at 44° for 15 min were comparable to the effects of 44° treatment alone for 30 min. The synergistic effect of cysteamine with the conditioning heat treatment at 44° was blocked by catalase (50 μg/mL). Following initial treatment with cysteamine at 37°, cells became thermotolerant within 2 h. The D0 of the survival curves for 44° heat treatments increased with duration (t1 = minutes, 37°) of the cysteamine (0.4 mM) exposure; e.g., the D0 increased by factors of 1.5, 1.6, 2.2, and 2.6 for t1 = 30, 60, 90, and 120 min, resp. The induction of thermotolerance by cysteamine at 37° was completely blocked by the addition of catalase (50 μg/mL), present during the initial period of drug treatment. Combined cysteamine and heat treatment at 44°, but also cysteamine exposure at 37°, enhanced synthesis of heat shock proteins. The data suggest that oxidative stress by cysteamine can be synergistic with the conditioning heat treatment at 44° which induces thermotolerance. At 37°, cysteamine itself induces thermotolerance and the enhanced synthesis of heat shock proteins under these conditions.

IT 60-23-1
RL: BIOL (Biological study)
(heat tolerance in animal cell in response to)

RN 60-23-1 CAPLUS

CN Ethanethiol, 2-amino- (8CI, 9CI) (CA INDEX NAME)

$H_2N-CH_2-CH_2-SH$

L4 ANSWER 23 OF 88 CAPLUS COPYRIGHT 2008 ACS on STN

ACCESSION NUMBER: 1987:509126 CAPLUS

DOCUMENT NUMBER: 107:109126

ORIGINAL REFERENCE NO.: 107:17571a,17574a

TITLE: Effect of pretreatment with cimetidine and cysteamine on increased vascular permeability and vascular injuries in cold-restraint rats

AUTHOR(S): Yabana, T.; Kondo, Y.; Kobayashi, T.; Narasaki, Y.; Yachi, A.

CORPORATE SOURCE: Dep. Intern. Med., Sapporo Med. Coll., Sapporo, Japan

SOURCE: New Trends Peptic Ulcer Chronic Hepatitis, Proc. Int. Symp. Jpn. Soc. Gastroenterol. (1987), Meeting Date 1985, Volume 1, 181-8. Excerpta Med.: Tokyo, Japan.
CODEN: 55YJA9

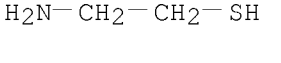
DOCUMENT TYPE: Conference

LANGUAGE: English

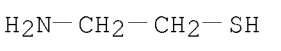
AB Cimetidine and cysteamine protected gastric mucosa from vascular permeability and vascular injury induced by cold-restraint stress in rats.

IT 60-23-1, Cysteamine

RL: BIOL (Biological study)
(stress-induced vascular injury and permeability in stomach response to)
RN 60-23-1 CAPLUS
CN Ethanethiol, 2-amino- (8CI, 9CI) (CA INDEX NAME)



L4 ANSWER 24 OF 88 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1986:143398 CAPLUS
DOCUMENT NUMBER: 104:143398
ORIGINAL REFERENCE NO.: 104:22551a,22554a
TITLE: Inhibition of liver microsomal calcium ion sequestration by oxidative stress: role of protein sulfhydryl groups
AUTHOR(S): Bellomo, G.; Richelmi, P.; Mirabelli, F.; Marinoni, V.; Abbagnano, A.
CORPORATE SOURCE: Dip. Med. Intern. Ter. Med., Univ. Pavia, Pavia, 27100, Italy
SOURCE: Free Radicals Liver Inj., Proc. Int. Meet., 1st (1985), 139-42. Editor(s): Poli, Giuseppe. IRL: Oxford, UK.
CODEN: 54ZCAK
DOCUMENT TYPE: Conference
LANGUAGE: English
AB Of 5 different disulfides tested for their ability to inhibit Ca uptake by liver microsomes cystamine [51-85-4] was the most potent. This effect was dependent on the cystamine concentration and the ability of cytamine to form
mixed disulfides with microsomal proteins. Glutathione [70-18-8] and dithiothreitol [3483-12-3] prevented cystamine-induced mixed disulfide formation and Ca transport inhibition. Cysteamine [60-23-1] also counteracted the inhibitory effect of cystamine.
IT 60-23-1
RL: BIOL (Biological study)
(calcium of uptake by liver microsomes inhibition by disulfides response to, protein SH groups in relation to)
RN 60-23-1 CAPLUS
CN Ethanethiol, 2-amino- (8CI, 9CI) (CA INDEX NAME)



L4 ANSWER 25 OF 88 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1986:15371 CAPLUS
DOCUMENT NUMBER: 104:15371
ORIGINAL REFERENCE NO.: 104:2517a,2520a
TITLE: Cysteamine effects on monoamines, dopamine-β-hydroxylase and the hypothalamic-pituitary axis
AUTHOR(S): Terry, L. C.; Craig, R.
CORPORATE SOURCE: Dep. Neurol. Physiol., Univ. Michigan, Ann Arbor, MI, 48105, USA
SOURCE: Neuroendocrinology (1985), 41(6), 467-75
CODEN: NUNDAJ; ISSN: 0028-3835
DOCUMENT TYPE: Journal

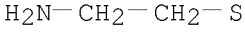
LANGUAGE: English

AB Rats were administered cysteamine (MEA) [60-23-1] (75-300 mg/kg, s.c.) and hypothalamic levels of dopamine (DA) [51-61-6], norepinephrine (NE) [51-41-2], epinephrine (EPI) [51-43-4], 5-HT, and MEA were measured by HPLC with electrochem. detection. Dopamine-β-hydroxylase (DBH) [9013-38-1] activity was measured in vitro after exposure to MEA with and without N-ethylmaleimide (NEMI). Chronically cannulated rats were administered MEA (100 or 300 mg/kg) and serial blood samples were removed in undisturbed animals, and after 30 min swimming stress. Cannulated rats with bilateral lesions of the ventromedial/arcuate nuclei (VMN/ARC) were administered MEA (150 mg/kg). MEA caused a dose-related decrease in hypothalamic NE and EPI and increased DA at doses ≤150 mg/kg. Tissue MEA was highest at 4 h (679 pM/mg tissue), but still measureable after 24 h. MEA inhibited DBH in vitro (95% inhibition at 10-3M); NEMI blocked inhibition. Stress-induced GH suppression and corticosterone [50-22-6] release were partially blocked by a low dose of MEA (100 mg/kg). Immediately after stress, plasma levels of growth hormone (GH) [9002-72-6] and TSH [9002-71-5] increased but this response was blocked by a high dose of MEA (300 mg/kg). MEA increased basal GH levels, but did not restore episodic GH secretion, and lowered prolactin (PRL) [9002-62-4] levels in VMN/ARC-lesioned animals. Apparently: (1) NE and EPI facilitate episodic GH and TSH release, (2) SRIF [51110-01-1] maintains low basal levels of GH and TSH, (3) MEA-induced PRL depletion does not involve DA systems and (4) MEA can be measured in tissue relatively simply by using HPLC with electrochem. detection.

IT 60-23-1
RL: BIOL (Biological study)
(somatotropin and TSH secretion inhibition by, mechanism of)

RN 60-23-1 CAPLUS

CN Ethanethiol, 2-amino- (8CI, 9CI) (CA INDEX NAME)



L4 ANSWER 26 OF 88 CAPLUS COPYRIGHT 2008 ACS on STN

ACCESSION NUMBER: 1972:81261 CAPLUS

DOCUMENT NUMBER: 76:81261

ORIGINAL REFERENCE NO.: 76:13037a,13040a

TITLE: Effect of radioprotectors from a group of aminothiols on the function of guinea pig heart under overloading conditions

AUTHOR(S): Kozlov, V. A.; Davydov, B. I.

CORPORATE SOURCE: USSR

SOURCE: Problemy Kosmicheskoi Biologii (1971), 14, 33-7
CODEN: PKBBA7; ISSN: 0555-2788

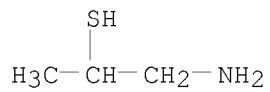
DOCUMENT TYPE: Journal

LANGUAGE: Russian

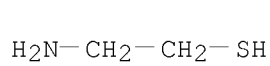
AB Cystamine [51-85-4] (150 mg/kg i.p.) and β-mercaptopropylamine [598-36-7] (150 mg/kg i.p.) caused a 23-50% slowing of the heart rate of guinea pigs and caused a number of other electrocardiograph changes. AET (I) [56-10-0] at 100-150 mg/kg i.p. had no effect on heart rate. All three compds. lowered the resistance of the animals to centrifugal stress and decreased their heart rate during the stress.

IT 598-36-7
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(heart response to, stress in relation to)

RN 598-36-7 CAPLUS
CN 2-Propanethiol, 1-amino- (6CI, 8CI, 9CI) (CA INDEX NAME)



L4 ANSWER 27 OF 88 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2005:327547 CAPLUS
DOCUMENT NUMBER: 143:222358
TITLE: Effects of cysteamine on performance of late-lactation cows during hot summer
AUTHOR(S): Shen, Zanming; Zhang, Rongfei
CORPORATE SOURCE: Lab of Animal Physiology and Biochemistry, Nanjing Agricultural University, Nanjing, Jiangsu Province, 210095, Peop. Rep. China
SOURCE: Zhongguo Yingyong Shenglixue Zazhi (2004), 20(4), 402-405
CODEN: ZYSZE2; ISSN: 1000-6834
PUBLISHER: Zhongguo Yingyong Shenglixue Zazhi Bianjibu
DOCUMENT TYPE: Journal
LANGUAGE: Chinese
AB In this experiment 96 black and white dairy cows, based on milk yield (M) prior to the experiment, were assigned into 4 groups (G): G1 (M<24 kg/d), G2 (24<M<28 kg/d), G3 (28<M<32 kg/d) and G4 (M>32 kg/d). Each group (n=24) was further divided into subgroups of Lactonin (3000 U/d) treatment (LT, n=49) and control (n=47). In G1 of LT, the rectal temperature decreased (P<0.05), milk yield, fat-corrected milk, milk fat and feed conversion rate (FCR) increased (P<0.05). These were accompanied with trendy of higher milk protein and lower somatic cell count. With whole LT cows (n=49), the mean milk fat (%) increased (P<0.05), mean milk protein tended to increase, and the mean milk yield and FCM tended to be enhanced. Plasma T3, T4 tended to decline whereas insulin enhanced (P<0.01) significantly in LT herd (n=49). Lactonin helps heat-stressed cow to maintain more normal metabolism in hot summer. This pos. effect of Lactonin on cow performance is associated with Lactonin-dependent alteration of plasma insulin, T3 and T4.
IT 60-23-1, Cysteamine
RL: FFD (Food or feed use); PAC (Pharmacological activity); BIOL (Biological study); USES (Uses)
(effects of cysteamine on performance of late-lactation cows during hot summer)
RN 60-23-1 CAPLUS
CN Ethanethiol, 2-amino- (8CI, 9CI) (CA INDEX NAME)



L4 ANSWER 28 OF 88 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1997:181629 CAPLUS
DOCUMENT NUMBER: 126:221075
ORIGINAL REFERENCE NO.: 126:42643a,42646a
TITLE: Method for stimulating the immune system using a prolactin agonist
INVENTOR(S): Bernton, Edward W.; Holaday, John W.; Bryant, Henry U.
PATENT ASSIGNEE(S): Entremed, Inc., USA
SOURCE: U.S., 26 pp., Cont. of U.S. Ser. No. 161,905,

abandoned.
CODEN: USXXAM
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 5605885	A	19970225	US 1994-315199	19940929 <--

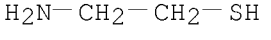
PRIORITY APPLN. INFO.:

US 1988-190568	B2	19880505
US 1990-586608	B1	19900924
US 1992-985434	B3	19921203
US 1993-161905	B1	19931203

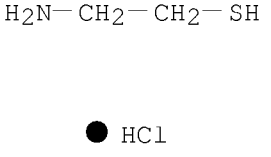
AB The present invention includes methods and compns. for affecting the immune system in animals and humans. The methods and compns. include the administration of prolactin agonists to an immunosuppressed animal or human thereby stimulating the immune system. The compds. that have prolactin-like activity include, but are not limited to, prolactin, prolactin peptide sequences, growth hormone, growth hormone peptide sequences, and any genetically engineered protein sequence with prolactin-like activity. The compds. of the invention can be used to antagonize suppression of the immune system by chronic stress, glucocorticosteroid therapy, radiation, chemotherapy, etc. In addition, the present invention includes a vaccine adjuvant comprising the administration of a prolactin agonist with the vaccine.

IT 60-23-1, Cysteamine 156-57-0, Cysteamine hydrochloride
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(method for stimulating immune system using a prolactin agonist)

RN 60-23-1 CAPLUS
CN Ethanethiol, 2-amino- (8CI, 9CI) (CA INDEX NAME)

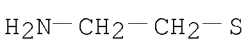


RN 156-57-0 CAPLUS
CN Ethanethiol, 2-amino-, hydrochloride (1:1) (CA INDEX NAME)



L4 ANSWER 29 OF 88 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1983:101537 CAPLUS
DOCUMENT NUMBER: 98:101537
ORIGINAL REFERENCE NO.: 98:15365a,15368a
TITLE: The effects of cysteamine on thyrotropin and immunoreactive β-endorphin secretion in the rat
AUTHOR(S): Millard, William J.; Sagar, Stephen M.; Badger, Thomas M.; Carr, Daniel B.; Arnold, Michael A.; Spindel, Eliot; Kasting, Norman W.; Martin, Joseph B.
CORPORATE SOURCE: Dep. Neurol., Massachusetts Gen. Hosp., Boston, MA, 02114, USA

SOURCE: Endocrinology (1983), 112(2), 518-25
CODEN: ENDOAO; ISSN: 0013-7227
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The effects of cysteamine (CSH) [60-23-1] on physiol. TSH [9002-71-5] and β-endorphin [60617-12-1] secretion were studied in the adult male rat. CSH at 90 and 300 mg/kg decreased plasma TSH, whereas at 30 mg/kg it did not alter plasma TSH levels. After the higher doses of CSH, TSH levels in the blood remained lower than control values on day 2, but returned to normal by 1 wk. This decrease in TSH within the plasma was not associated with a reduction in hypothalamic TRH concns. The TSH response to 500 ng/kg TRH was normal in CSH-treated animals. Blockade of norepinephrine [51-41-2] synthesis with diethyldithiocarbamate (500 mg/kg) or fusaric acid (100 mg/kg) inhibited TSH secretion in a manner similar to that of CSH. β-Endorphin-like immunoreactivity (β-End-LI) was elevated in the plasma immediately after CSH (300 mg/kg) administration. This was associated with a 58% reduction in anterior pituitary β-End-LI and no change in hypothalmic β-End-LI. Plasma β-End-LI returned to normal on day 2. The increase in plasma β-End-LI induced by immobilization stress was not compromised by CSH treatment. The observed effects of CSH on both TSH and β-End-LI are consistent with a reduction in central norepinephrine neurotransmission through the known action of CSH to inhibit dopamine β-hydroxylase. Acute stress may play a role as well in the observed changes in TSH and β-End-LI secretion.
IT 60-23-1
RL: BIOL (Biological study)
(endorphin and TSH secretion response to, central noradrenergic neurotransmission in relation to)
RN 60-23-1 CAPLUS
CN Ethanethiol, 2-amino- (8CI, 9CI) (CA INDEX NAME)



L4 ANSWER 30 OF 88 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2000:736046 CAPLUS
DOCUMENT NUMBER: 134:28250
TITLE: Pantetheinase activity of membrane-bound Vanin-1: lack of free cysteamine in tissues of Vanin-1 deficient mice
AUTHOR(S): Pitari, G.; Malergue, F.; Martin, F.; Philippe, J. M.; Massucci, M. T.; Chabret, C.; Maras, B.; Dupre, S.; Naquet, P.; Galland, F.
CORPORATE SOURCE: Dipartimento di Biologia di Base ed Applicata Universita' di L'Aquila, L'Aquila, Coppito, 67010, Italy
SOURCE: FEBS Letters (2000), 483(2,3), 149-154
CODEN: FEBLAL; ISSN: 0014-5793
PUBLISHER: Elsevier Science B.V.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Pantetheinase (EC 3.5.1.-) is an ubiquitous enzyme which in vitro has been shown to recycle pantothenic acid (vitamin B5) and to produce cysteamine, a potent anti-oxidant. We show that the Vanin-1 gene encodes pantetheinase widely expressed in mouse tissues: (1) a pantetheinase activity is specifically expressed by Vanin-1 transfectants and is immunodepleted by specific antibodies; (2) Vanin-1 is a GPI-anchored

pantetheinase, and consequently an ectoenzyme; (3) Vanin-1 null mice are deficient in membrane-bound pantetheinase activity in kidney and liver; (4) in these organs, a major metabolic consequence is the absence of detectable free cysteamine; this demonstrates that membrane-bound pantetheinase is the main source of cysteamine in tissues under physiol. conditions. Since the Vanin-1 mol. was previously shown to be involved in the control of thymus reconstitution following sublethal irradiation in vivo, this raises the possibility that Vanin/pantetheinase might be involved in the regulation of some immune functions maybe in the context of the response to oxidative stress.

IT 60-23-1, Cysteamine
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(pantetheinase activity directly correlates the tissues level of free cysteamine in mice)

RN 60-23-1 CAPLUS
CN Ethanethiol, 2-amino- (8CI, 9CI) (CA INDEX NAME)

$H_2N-CH_2-CH_2-SH$

REFERENCE COUNT: 33 THERE ARE 33 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 31 OF 88 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1997:718837 CAPLUS
DOCUMENT NUMBER: 128:7207
ORIGINAL REFERENCE NO.: 128:1383a,1386a
TITLE: Reduction of human hair by cysteamine and ammonium thioglycolate: a correlation of amino acid analysis and single-fiber tensile kinetic data
AUTHOR(S): Manuszak, Melissa A.; Borish, Edward T.; Wickett, R. Randall
CORPORATE SOURCE: College Pharmacy, Univ. Cincinnati, Cincinnati, OH, 45267, USA
SOURCE: Journal of the Society of Cosmetic Chemists (1996), 47(4), 213-227
CODEN: JSCCA5; ISSN: 0037-9832
PUBLISHER: Society of Cosmetic Chemists
DOCUMENT TYPE: Journal
LANGUAGE: English

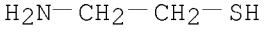
AB A study was conducted to determine the effects of reduction by cysteamine and ammonium thioglycolate (ATG) on the phys. and chemical properties of human hair. The methods utilized were amino acid anal. with ortho-phthalaldehyde derivatization (OPA) and a modification of the single-fiber tensile kinetics (SFTK) method. Virgin, medium brown hair from a single source (DeMeo Brothers) was used for all of the expts. Stress relaxation of hair fibers was monitored to determine the rate of reduction of stress-supporting disulfide bonds by cysteamine and ATG. Levels of cystine and cysteine were monitored by amino acid anal. to determine the rate of reduction of disulfide bonds in the whole fiber. The results of this study indicated that the rate of reduction of both stress -supporting and whole-fiber disulfide bonds by ammonium thioglycolate was faster than the rate of reduction by cysteamine. The kinetic results obtained by stress relaxation were found to agree with the results from amino acid anal.

IT 60-23-1, Cysteamine
RL: BUU (Biological use, unclassified); RCT (Reactant); BIOL (Biological study); RACT (Reactant or reagent); USES (Uses)

(correlation of amino acid anal. and single-fiber tensile kinetic data in reduction of human hair by cysteamine and ammonium thioglycolate)
RN 60-23-1 CAPLUS
CN Ethanethiol, 2-amino- (8CI, 9CI) (CA INDEX NAME)

REFERENCE COUNT: 40 THERE ARE 40 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 32 OF 88 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1983:101536 CAPLUS
DOCUMENT NUMBER: 98:101536
ORIGINAL REFERENCE NO.: 98:15365a,15368a
TITLE: Cysteamine effects on growth hormone secretion in the male rat
AUTHOR(S): Millard, William J.; Sagar, Stephen M.; Badger, Thomas M.; Martin, Joseph B.
CORPORATE SOURCE: Dep. Gynecol., Massachusetts Gen. Hosp., Boston, MA, 02114, USA
SOURCE: Endocrinology (1983), 112(2), 509-17
CODEN: ENDOAO; ISSN: 0013-7227
DOCUMENT TYPE: Journal
LANGUAGE: English
AB cysteamine (CSH) [60-23-1] at 3.0, 9.0, 30.0, 90.0 or 300 mg/kg altered physiol. growth hormone (GH) [9002-72-6] secretion and this effect was dose-dependent and reversible. Lower doses appeared to have a specific effect on immunoreactive somatostatin (SS) [51110-01-1]-mediated inhibition of GH secretion. On the other hand, high doses of CSH totally disrupted GH secretion by affecting both the SS inhibitory and the GH-releasing factor stimulatory components of episodic GH secretion. The latter action of CSH is, perhaps, mediated by both an inhibition of the synthesis of norepinephrine [51-41-2] in the hypothalamus and an acute stress response.
IT 60-23-1
RL: BIOL (Biological study)
(growth hormone secretion response to)
RN 60-23-1 CAPLUS
CN Ethanethiol, 2-amino- (8CI, 9CI) (CA INDEX NAME)



L4 ANSWER 33 OF 88 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1989:632106 CAPLUS
DOCUMENT NUMBER: 111:232106
ORIGINAL REFERENCE NO.: 111:38545a,38548a
TITLE: Alkylamides (e.g. cysteamine derivatives) as peptic ulcer inhibitors
INVENTOR(S): Iwai, Masakazu; Kohda, Isao; Fukaya, Chikara; Arakawa, Yoshio
PATENT ASSIGNEE(S): Green Cross Corp., Japan
SOURCE: Eur. Pat. Appl., 11 pp.
CODEN: EPXXDW
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1

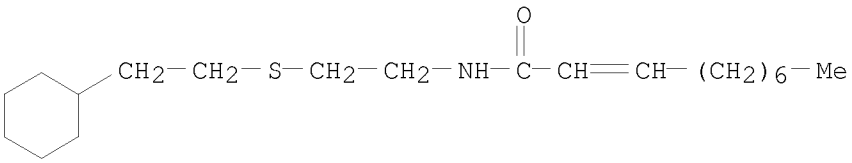
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
EP 319644	A2	19890614	EP 1988-110388	19880629 <--
EP 319644	A3	19900613		
R: BE, CH, DE, ES, FR, GB, IT, LI, NL, SE				
JP 02000269	A	19900105	JP 1988-81104	19880331 <--

PRIORITY APPLN. INFO.: JP 1987-282747 A 19871109
OTHER SOURCE(S): MARPAT 111:232106

AB Alkylamides, e.g. Me(CH2)mCH:CH(CH2)nCONHAYX [m + n = 0-14 preferably 6,7; m = 3-14 preferably 6,7; n = 0, 1 preferably O; A = alkylene, preferably (CH2)2,CH2; Y = S, SO; X = alkyl, cycloalkyl, aralkyl, N-alkyl (-substituted) piperidinylalkyl, N,N-dialkylcarboxamidoalkyl] are prepared Amidation of cysteamine with 2-decenoyl chloride (preparation given) in EtOAc in the presence of Et3N gave N,N'-bis(2-decenoyl)cysteamine, which in MeOH-H2O was successively treated with Bu3P and (2-iodoethyl)cyclohexane to afford S-cyclohexylethyl-N-(2-decenyl)cysteamine. The latter at 5 mg/kg showed 69.5% inhibition of H2O-stress-induced ulcer in rats.

IT 123911-82-0P
RL: SPN (Synthetic preparation); PREP (Preparation)
(preparation of, as peptic ulcer inhibitor)
RN 123911-82-0 CAPLUS
CN 2-Decenamide, N-[2-[(2-cyclohexylethyl)thio]ethyl]- (CA INDEX NAME)

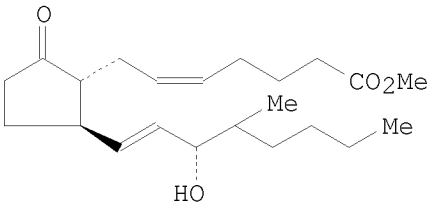


IT 60-23-1, Cysteamine
RL: RCT (Reactant); RACT (Reactant or reagent)
(reaction of, in preparation of peptic ulcer inhibitors)
RN 60-23-1 CAPLUS
CN Ethanethiol, 2-amino- (8CI, 9CI) (CA INDEX NAME)

L4 ANSWER 34 OF 88 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1995:985129 CAPLUS
DOCUMENT NUMBER: 124:21765
ORIGINAL REFERENCE NO.: 124:3991a,3994a
TITLE: Effect of cysteamine on glutathione level and developmental capacity of bovine oocyte matured in vitro
AUTHOR(S): de Matos, Daniel G.; Furnus, Cecilia C.; Moses, Daniel F.; Baldassarre, Hernan
CORPORATE SOURCE: Fundacion Margarita Perez Companc, Centro de Investigaciones Reproductivas Perez Companc, Buenos Aires, Argent.
SOURCE: Molecular Reproduction and Development (1995), 42(4), 432-6
CODEN: MREDEE; ISSN: 1040-452X

PUBLISHER: Wiley-Liss
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The present study was carried out to evaluate if the addition of cysteamine to the culture medium during in vitro maturation of bovine oocytes increased the glutathione (GSH) levels in the mature oocytes, and if these changes may promote an improvement in vitro development to the blastocyst stage. Follicular oocytes from slaughterhouse ovaries were matured in TCM 199 supplemented with 10% (volume/volume) fetal calf serum, hormones, and 0 (control), 25, 50, or 100 μM of cysteamine for 24 h. After in vitro maturation the oocytes were fertilized and cultured for 8 days. The percentage of embryos that developed to the blastocyst stage was significantly higher ($P<0.01$) for ooxytes matured in medium containing 100 μM of cysteamine than for those matured in control medium. Moreover, the intracellular GSH levels were increased ($P<0.05$) in oocytes matured with 100 μM of cysteamine with respect to control. No differences were observed in maturation and cleavage rates, and in the mean cell nos. per blastocyst among treatments ($P<0.05$). These results indicate that the addition of thiol compds. such as cysteamine to maturation medium increases the efficiency of in vitro blastocyst production from immature bovine oocytes. The higher levels of GSH in oocytes matured in the presence of cysteamine suggest that the beneficial effects of cysteamine on in vitro maturation and subsequent development after in vitro fertilization are mediated by GSH.
IT 60-23-1, Cysteamine
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(cysteamine effect on glutathione level and developmental capacity of bovine oocyte matured in vitro)
RN 60-23-1 CAPLUS
CN Ethanethiol, 2-amino- (8CI, 9CI) (CA INDEX NAME)

L4 ANSWER 35 OF 88 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1987:188784 CAPLUS
DOCUMENT NUMBER: 106:188784
ORIGINAL REFERENCE NO.: 106:30457a,30460a
TITLE: Gastrointestinal activity of a new antiulcer: FCE 20700 (11-deoxy-13,14-didehydro-16(S)-methyl PGE2 methylester)
AUTHOR(S): Arrigoni, C.; Ceserani, R.; Mizzotti, B.; Ferrari, M.; Soldani, G.; Costa, G.
CORPORATE SOURCE: Ric. e Sviluppo, Farmitalia Carlo Erba, Milan, Italy
SOURCE: Adv. Pharmacol. Res. Pract., Proc. Congr. Hung. Pharmacol. Soc., 4th (1986), Meeting Date 1985, Volume 3, 481-7. Editor(s): Knoll, Jozsef; Kelemen, Karoly. Pergamon: Oxford, UK.
CODEN: 55NPA6
DOCUMENT TYPE: Conference
LANGUAGE: English
GI



I

AB In the rat, oral FCE 20700 (I) [89648-76-0] prevents gastric ulcers induced by stress (ED50 = 148 μg/kg), ethanol [64-17-5] (ED50 = 9 μg/kg), or indomethacin [53-86-1] (ED50 = 38 μg/kg), duodenal ulcers induced by cysteamine [60-23-1] (ED50 = 191 μg/kg), and intestinal ulcers induced by indomethacin (ED50 = 557 μg/kg). It has a weak effect on rat basal gastric acid secretion (ED50 = 2385 μg/kg), thus displaying clear-cut cytoprotective activity. In the conscious dog with gastric fistula and Heidenhain pouch, intragastric I weakly inhibits gastric acid secretion stimulated by pentagastrin [5534-95-2] and histamine [51-45-6]. It induces diarrhea in rats at 6250 μg/kg p.o. I at 5 μg/kg s.c. prevents rat hepatotoxicity induced by CCl4 [56-23-5], as judged by serum glutamate-pyruvate transaminase and diazepam plasma concentration I does not interfere with the antiinflammatory activity of indomethacin, thus allowing use of higher doses of indomethacin without enhancement of its gastrointestinal side-effects when it is combined with I.

IT 60-23-1, Cysteamine
    RL: BIOL (Biological study)
    (ulcer induction by, FCE 20700 inhibition of)

RN 60-23-1 CAPLUS

CN Ethanethiol, 2-amino- (8CI, 9CI) (CA INDEX NAME)

$H_2N-CH_2-CH_2-SH$

L4 ANSWER 36 OF 88 CAPLUS COPYRIGHT 2008 ACS on STN

ACCESSION NUMBER: 1990:116414 CAPLUS

DOCUMENT NUMBER: 112:116414

ORIGINAL REFERENCE NO.: 112:19666h,19667a

TITLE: Cellular recovery of glyceraldehyde-3-phosphate dehydrogenase activity and thiol status after exposure to hydroperoxides

AUTHOR(S): Brodie, Ann E.; Reed, Donald J.

CORPORATE SOURCE: Dep. Biochem. Biophys., Oregon State Univ., Corvallis, OR, 97331-6503, USA

SOURCE: Archives of Biochemistry and Biophysics (1990), 276(1), 212-18
CODEN: ABBIA4; ISSN: 0003-9861

DOCUMENT TYPE: Journal

LANGUAGE: English

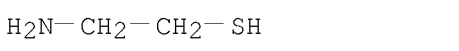
AB The activity of the thiol-dependent enzyme glyceraldehyde 3-phosphate dehydrogenase (GPD), in vertebrate cells, was modulated by a change in the intracellular thiol:disulfide redox status. Human lung carcinoma cells (A549) were incubated with 1-120 mM H2O2, 1-120 mM tert-Bu hydroperixode, 1-6 mM ethacrynic acid, or 0.1-10 mM N-ethylmaleimide for 5 min. Loss of reduced protein thiols, as measured by binding of the thiol reagent iodoacetic acid to GPD, and loss of GDP enzymic activity occurred in a dose-dependent manner. Incubation of the cells, following oxidative treatment, in saline for 30 min or with 20 mM DTT partially reversed both

changes in GPD. The enzymic recovery of GDP activity was observed either without addition of thiols to the medium or by incubation of a sonicated cell mixture with 2 mM cysteine, cystine, cysteamine, or GSH; GSSG had no effect. Treatment of cells with buthione sulfoximine to decreased cellular GSH by varying amts. caused a dose-related increase in sensitivity of GPD activity to in activation by H2O2 and decreased cellular ability for subsequent recovery. GPD responded in a similar fashion with oxidation treatment of another lung carcinoma cell line (A427) as well as normal lung tissue from human and rat. Apparently, the cellular thiol redox status can be important in determining GPD enzymic activity.

IT 60-23-1P, Cysteamine
RL: BIOL (Biological study); PREP (Preparation)
(glyceraldehyde phosphate dehydrogenase recovery from exposure to hydroperoxides enhancement by)

RN 60-23-1 CAPLUS

CN Ethanethiol, 2-amino- (8CI, 9CI) (CA INDEX NAME)



L4 ANSWER 37 OF 88 CAPLUS COPYRIGHT 2008 ACS on STN

ACCESSION NUMBER: 1987:44561 CAPLUS

DOCUMENT NUMBER: 106:44561

ORIGINAL REFERENCE NO.: 106:7293a,7296a

TITLE: Gastric antisecretory and antiulcer properties of enprostil, (±)-11α,15α-dihydroxy-16-phenoxy-17,18,19,20-tetranor-9-oxoprosta-4,5,13(t)-trienoic acid methyl ester

AUTHOR(S): Roszkowski, A. P.; Garay, G. L.; Baker, S.; Schuler, M.; Carter, H.

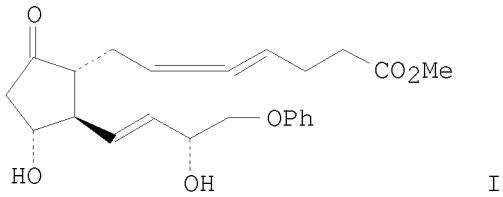
CORPORATE SOURCE: Inst. Pharmacol. Metab., Syntex Res., Palo Alto, CA, 94304, USA

SOURCE: Journal of Pharmacology and Experimental Therapeutics (1986), 239(2), 382-9
CODEN: JPETAB; ISSN: 0022-3565

DOCUMENT TYPE: Journal

LANGUAGE: English

GI



I

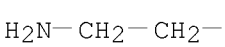
AB In rats, in which both the pylorus and esophagus were ligated, oral half-maximum EDs (ED50) for enprostil (I) [82444-04-0] for inhibiting acid secretion evoked by histamine [51-45-6], pentagastrin [5534-95-2], and carbachol [51-83-2] were 9.9, 40, and 0.83 μg/kg, resp. In inhibiting histamine-evoked acid secretion, I was more potent when administered orally than when injected into the duodenum or s.c. When I was injected directly into the pouch of Heidenhain dogs, intense antisecretory activity

occurred, ED50 = 0.9 μg/kg, whereas, when given orally to the main stomach the ED50 was 6.6 μg/kg. Administration of cimetidine either orally or to the pouch resulted in virtually identical ED50 values, 2.9 and 3.1 mg/kg. I also inhibited dimaprit [65119-89-3]- and pentagastrin [5534-95-2]-induced acid secretion in cats with permanent gastric fistulae. The oral ED50 values for inhibiting acid secretion evoked by these 2 secretagogues were 2.5 and 0.8 μg/kg, resp. I was extremely potent in preventing indomethacin [53-86-1] plus cold stress ulcers in rats. When given orally the ED50 was 0l61 and s.c. it was 22 μg/kg. It was also highly potent in preventing cysteamine [60-23-1]-induced duodenal ulcers when given orally, ED50 = 20 μg/kg. Thus, I is a highly potent antisecretory and antiulcer agent. It appears to act topically; directly at gastric mucosal sites.

IT 60-23-1, Cysteamine
RL: BIOL (Biological study)
(duodenal ulcer formation in response to, enprostil inhibition of)

RN 60-23-1 CAPLUS

CN Ethanethiol, 2-amino- (8CI, 9CI) (CA INDEX NAME)



L4 ANSWER 38 OF 88 CAPLUS COPYRIGHT 2008 ACS on STN

ACCESSION NUMBER: 2001:781247 CAPLUS

DOCUMENT NUMBER: 135:327326

TITLE: Method for identifying regulators of protein-advanced glycation end product (protein-AGE) formation

INVENTOR(S): Jacobson, Elaine L.; Jacobson, Myron K.; Wondrak, Georg Thomas

PATENT ASSIGNEE(S): Niadyne Corporation, USA; University of Kentucky

SOURCE: PCT Int. Appl., 50 pp.
CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001079842	A2	20011025	WO 2001-US12368	20010416 <--
WO 2001079842	A3	20021024		
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
US 20020037496	A1	20020328	US 2001-836576	20010416 <--
US 6716635	B2	20040406		
EP 1272843	A2	20030108	EP 2001-927070	20010416 <--
EP 1272843	B1	20070620		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
JP 2003531376	T	20031021	JP 2001-576457	20010416 <--
JP 3920641	B2	20070530		
AU 2001253555	B2	20050106	AU 2001-253555	20010416
CN 1205480	C	20050601	CN 2001-810726	20010416

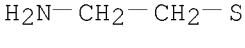
AT 365319	T	20070715	AT 2001-927070		20010416
ES 2286117	T3	20071201	ES 2001-927070		20010416
MX 2002PA10194	A	20040819	MX 2002-PA10194		20021014 <--
HK 1059117	A1	20050916	HK 2004-102069		20040322
PRIORITY APPLN. INFO.:			US 2000-197829P	P	20000414
			WO 2001-US12368	W	20010416

AB Methods are provided for identifying compds. which affect cellular stress. In particular, the method provides methods for identifying compds. which inhibit protein-advanced glycation end product formation, where the compds. are carbonyl scavengers which inhibit the formation. The assay involves combining the substance of interest with histone H1 and ADP-ribose, and then measuring fluorescence and protein crosslinking. Various inhibitors of protein-AGE glycation have been identified using this assay.

IT 60-23-1, Cysteamine
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(protein-advanced glycation end product formation regulator identification)

RN 60-23-1 CAPLUS

CN Ethanethiol, 2-amino- (8CI, 9CI) (CA INDEX NAME)



L4 ANSWER 39 OF 88 CAPLUS COPYRIGHT 2008 ACS on STN

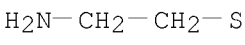
ACCESSION NUMBER: 1980:492770 CAPLUS
DOCUMENT NUMBER: 93:92770
ORIGINAL REFERENCE NO.: 93:14847a,14850a
TITLE: The regulation and function of taurine in the heart and other organs
AUTHOR(S): Huxtable, Ryan J.
CORPORATE SOURCE: Health Sci. Cent., Univ. Arizona, Tucson, AZ, 85724, USA
SOURCE: Nat. Sulfur Compd., [Proc. Int. Meet.], 3rd (1980), Meeting Date 1979, 277-93. Editor(s): Cavallini, Doriano; Gaull, Gerald E.; Zappia, Vincenzo. Plenum: New York, N. Y.
CODEN: 43SYAX
DOCUMENT TYPE: Conference
LANGUAGE: English

AB A discussion is given of the biosynthesis, transport, and possible function of taurine in the heart, as well as other organs, and the use of guanidinoethylsulfonate to inhibit taurine transport and to lower taurine content. The role of cysteamine in taurine formation in the heart and the effects of adrenergic stress on taurine transport and formation are discussed.

IT 60-23-1
RL: BIOL (Biological study)
(taurine formation from, by heart)

RN 60-23-1 CAPLUS

CN Ethanethiol, 2-amino- (8CI, 9CI) (CA INDEX NAME)



L4 ANSWER 40 OF 88 CAPLUS COPYRIGHT 2008 ACS on STN

ACCESSION NUMBER: 1956:74382 CAPLUS
DOCUMENT NUMBER: 50:74382
ORIGINAL REFERENCE NO.: 50:14027f-g
TITLE: The acetylating power of the liver of x-irradiated rats
AUTHOR(S): Koch, R.; Hagen, U.
CORPORATE SOURCE: Univ. Freiburg, i. Br., Germany
SOURCE: Proceedings of the International Congress of Biochemistry (1955) 121
CODEN: 18USAR
DOCUMENT TYPE: Journal
LANGUAGE: German

AB cf. C.A. 50, 6528g, 6656i, 9456i. Previous work with cysteamine (I) is reviewed. The acetylating power (II) of the liver of rats as a function of coenzyme A decreased by 50% 12 hrs. after x-irradiation (dosage not stated). This decrease in II was not prevented by pretreatment with I. A 4-day pretreatment of rats with 50 g./day pantothenic acid likewise did not prevent a fall in II (which declined 75%, 12 hrs. after 500 r.). Any protective action of I was not connected with any relation to coenzyme A. Decreased II was not a specific result of radiations; chilling (stress) likewise decreased it.

IT 60-23-1, Ethanethiol, 2-amino-
(effect on acetyl in liver after x-ray treatment)
RN 60-23-1 CAPLUS
CN Ethanethiol, 2-amino- (8CI, 9CI) (CA INDEX NAME)

L4 ANSWER 41 OF 88 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2003:796656 CAPLUS
DOCUMENT NUMBER: 139:307549
TITLE: Preparation of cyclopentanones and cyclohexanones for use in pharmaceutical compositions
INVENTOR(S): Roberts, Stanley Michael; Ross, Nicolette Christa; Jadhav, Vasudev; Evans, Paul; Snape, Timothy James; Happe, Alan Michael; Santoro, Gabriella Maria
PATENT ASSIGNEE(S): Charterhouse Therapeutics Limited, UK
SOURCE: PCT Int. Appl., 139 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 3
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003082813	A2	20031009	WO 2003-GB1379	20030327 <--
WO 2003082813	A3	20040122		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

WO 2003051807 A2 20030626 WO 2002-GB5708 20021216 <--
WO 2003051807 A3 20030918
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG
WO 2003051893 A2 20030626 WO 2002-GB5709 20021216 <--
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG
CA 2480857 A1 20031009 CA 2003-2480857 20030327 <--
AU 2003224244 A1 20031013 AU 2003-224244 20030327 <--
EP 1487789 A2 20041222 EP 2003-720667 20030327 <--
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK
JP 2005521726 T 20050721 JP 2003-580281 20030327
IN 2004CN02121 A 20060303 IN 2004-CN2121 20040923
PRIORITY APPLN. INFO.: GB 2002-7232 A 20020327
WO 2002-GB5708 A 20021216
WO 2002-GB5709 A 20021216
GB 2001-29979 A 20011214
GB 2001-29980 A 20011214
WO 2003-GB1379 W 20030327

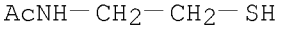
OTHER SOURCE(S): MARPAT 139:307549

AB Cyclopentanones, cyclopentenones, cyclohexanones or cyclohexenones substituted by -SR [R is an (un)substituted alk(en)(yn)yl, aryl, or aralk(en)(yn)yl group that may optionally include at least one heteroatom in its carbon skeleton] and possibly other groups were prepared for use in pharmaceutical compns. The compds. are either: (a) more soluble in water at 20-40°C, (b) less lipophilic, and/or (c) have a greater therapeutic index or (d) less soluble in water at 20-40°C, (e) more lipophilic, and/or (f) have a greater therapeutic index than an equivalent 2-cyclohexenone or 2-cyclopentenone derivative in which a hydrogen atom replaces the -SR group. Thus, (S)-4-(tert-butyldimethylsilyloxy)-2-cyclopentenone underwent addition reaction with thiols [p-tolyl mercaptan, methoxythiophenol, 4-pyridinethiol, Boc-L-Cys-OH, etc.] to afford cyclopentanone thioethers, which were assayed for effect on reactivity of transcription factors HSF and NF-κB or replication of Sendai virus, cytotoxicity, antiinflammatory effect, etc.

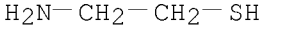
IT 1190-73-4, n 2 Mercaptoethyl acetamide
RL: RCT (Reactant); RACT (Reactant or reagent)
(preparation of cyclopentanone and cyclohexanone thioethers for use in pharmaceutical compns.)

RN 1190-73-4 CAPLUS

CN Acetamide, N-(2-mercaptoethyl)- (CA INDEX NAME)



L4 ANSWER 42 OF 88 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2003:728377 CAPLUS
DOCUMENT NUMBER: 139:290328
TITLE: Effect of cysteamine on redox-sensitive thiol-containing proteins in the duodenal mucosa
AUTHOR(S): Khomenko, Tetyana; Deng, Xiaoming; Jadus, Martin R.; Szabo, Sandor
CORPORATE SOURCE: Diagnostic and Molecular Medicine Health Care Group, Pathology and Laboratory Medicine Service, VA Medical Center, Long Beach, CA, 90822, USA
SOURCE: Biochemical and Biophysical Research Communications (2003), 309(4), 910-916
CODEN: BBRCA9; ISSN: 0006-291X
PUBLISHER: Elsevier Science
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Recent studies from our laboratory demonstrated that Egr-1 is upregulated in the rat duodenal mucosa during cysteamine-induced duodenal ulceration and that antisense egr-1 oligonucleotide aggravates the duodenal ulcers. This study was aimed to determine the effects of cysteamine on redox-sensitive Egr-1 transcriptional activity and on other thiol-containing proteins such as redox factor-1 (Ref-1) and thioredoxin (Trx). Here we demonstrate for the first time that cysteamine increases the expression and nuclear translocation of Egr-1, Ref-1, and Trx, and activates binding of Egr-1 to DNA. Moreover, we also show that Egr-1 forms a complex with other redox-sensitive transcription factors (e.g., AP-1, AP-2, NFATc, Sp1, PAX-5, MTF-1, c-Myb, and CREB) in rat duodenal mucosa and that cysteamine enhances the formation of these complexes. The antioxidant ebselen markedly elevated the nuclear Ref-1 expression and Egr-1/DNA binding, and decreased the ulcerogenic effect of cysteamine as did catalase. Thus, redox-sensitive signaling systems seem to play an important role in cysteamine-induced duodenal ulceration.
IT 60-23-1, Cysteamine
RL: ADV (Adverse effect, including toxicity); BSU (Biological study, unclassified); BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)
(redox-sensitive Egr-1, redox factor-1 and thioredoxin expression and nuclear translocation and Egr-1 DNA-binding activity in cysteamine-induced duodenal ulceration)
RN 60-23-1 CAPLUS
CN Ethanethiol, 2-amino- (8CI, 9CI) (CA INDEX NAME)



REFERENCE COUNT: 40 THERE ARE 40 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 43 OF 88 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2000:790299 CAPLUS
DOCUMENT NUMBER: 133:317567
TITLE: Glycine cleavage system inhibitors as potential antipsychotics
INVENTOR(S): Arlt, Michael; Bartoszyk, Gerd
PATENT ASSIGNEE(S): Merck Patent G.m.b.H., Germany
SOURCE: PCT Int. Appl., 15 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent

LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

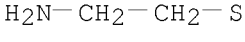
PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000066110	A1	20001109	WO 2000-EP3456	20000417 <--
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
CA 2372073	A1	20001109	CA 2000-2372073	20000417 <--
EP 1185259	A1	20020313	EP 2000-929364	20000417 <--
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
BR 2000010151	A	20020611	BR 2000-10151	20000417 <--
JP 2002543124	T	20021217	JP 2000-614995	20000417 <--
US 6395780	B1	20020528	US 2000-559831	20000428 <--
NO 2001005247	A	20011026	NO 2001-5247	20011026 <--
MX 2001PA10933	A	20020621	MX 2001-PA10933	20011026 <--

PRIORITY APPLN. INFO.:	US 1999-131647P	P	19990429
	EP 1999-108480	A	19990430
	WO 2000-EP3456	W	20000417

AB The invention relates to inhibitors of the glycine cleavage system and their use as potential antipsychotic agents. The invention relates furthermore to a process for treating humans having psychosis, psychosis associated with an illness, schizophrenia, Alzheimer's disease, or other related psychotic disorders.

IT 60-23-1, Cysteamine
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(glycine cleavage system inhibitor for antipsychotic)

RN 60-23-1 CAPLUS
CN Ethanethiol, 2-amino- (8CI, 9CI) (CA INDEX NAME)



REFERENCE COUNT: 10 THERE ARE 10 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

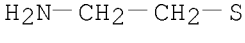
L4 ANSWER 44 OF 88 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1999:245544 CAPLUS
DOCUMENT NUMBER: 131:55890
TITLE: Antioxidants and biological radiation protection
AUTHOR(S): Lenton, K. J.; Greenstock, C. L.
CORPORATE SOURCE: Radiation Biology and Health Physics Branch, AECL, Chalk River Laboratories, Chalk River, ON, K0J 1J0, Can.
SOURCE: Annual Conference Proceedings - Canadian Nuclear Society (1998), 19th(Vol. 2), 5A1/1-5A1/7
CODEN: CCSCDZ; ISSN: 0227-1907
PUBLISHER: Canadian Nuclear Society
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Antioxidants and antioxidant enzymes, by combating oxygen radical-mediated radiation-induced oxidative stress, may prevent the accumulation of damage involved in tumor initiation, promotion and progression, and thus serve to protect us against ionizing radiation. We are testing the possible role of dietary antioxidants, and other biol. response modifiers, in determining individual radiation response. These expts. use the fluorescent protein beta-phycoerythrin as a target and biomol. marker for radiation-induced oxidative stress. Antioxidants are ranked according to their radioprotectiveness by their ability to compete with beta-phycoerythrin for radiolytic oxygen radicals. Samples of blood serum from cancer patients have been analyzed using this technique. There is a trend towards decreasing antioxidant levels with increasing donor age, and this is consistent with data showing an increasing radiosensitivity with age. We are presently monitoring antioxidant and antioxidant enzyme levels in atomic radiation workers and the general public, in order to assess whether they influence individual radiosensitivity. Knowledge of this source of biol. response modification will be useful in applying radiation protection practices to those individuals or groups most at risk, and for estimating individual risks associated with radiation exposure.

IT 60-23-1
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(use of β-phycoerythrin as biomarker for radiolytic free radicals in evaluating radioprotective action of antioxidants and other compds.)

RN 60-23-1 CAPLUS

CN Ethanethiol, 2-amino- (8CI, 9CI) (CA INDEX NAME)



REFERENCE COUNT: 8 THERE ARE 8 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 45 OF 88 CAPLUS COPYRIGHT 2008 ACS on STN

ACCESSION NUMBER: 1998:739931 CAPLUS

DOCUMENT NUMBER: 130:120350

TITLE: Thiol-Mediated Disassembly and Reassembly of [2Fe-2S] Clusters in the Redox-Regulated Transcription Factor SoxR

AUTHOR(S): Ding, Huangen; Demple, Bruce

CORPORATE SOURCE: Department of Cancer Cell Biology School of Public Health, Harvard University, Boston, MA, 02115-6021, USA

SOURCE: Biochemistry (1998), 37(49), 17280-17286
CODEN: BICHAW; ISSN: 0006-2960

PUBLISHER: American Chemical Society

DOCUMENT TYPE: Journal

LANGUAGE: English

AB SoxR, a transcription factor containing [2Fe-2S] clusters, governs the cellular response to oxidative stress in Escherichia coli. The oxidation state of the iron-sulfur clusters regulates the SoxR transcriptional activity. When the reduced iron-sulfur clusters become oxidized ([2Fe-2S]2+ state), SoxR is activated to stimulate transcription of the soxS gene, whose product in turn switches on a group of genes encoding various proteins that defend against oxidative stress and antibiotics. A previous study showed that the oxidized [2Fe-2S] clusters of SoxR are destroyed by a free-radical-dependent process in vitro during aerobic exposure to the biol. thiol glutathione. Here, we show that different thiols have differing effects on the SoxR [2Fe-2S] clusters. Like reduced glutathione, N-acetyl-L-cysteine, L-cysteine Me

ester, and L-cysteine Et ester disrupted the SoxR [2Fe-2S] clusters in aerobic solution This disruption was blocked by L-cysteine, which was effective at concns. 100-fold lower (1-10 μM) than the disrupting thiols (1 mM). In view of a previous observation that superoxide dismutase and catalase block the disruption process, this result suggests that L-cysteine may quench reactive SoxR or thiol intermediates involved in the cluster disruption reaction, the detailed mechanism of which remains unknown. In contrast, bifunctional thiols such as dithiothreitol or dithioerythritol promoted the aerobic assembly of the functional [2Fe-2S] clusters into apo-SoxR in the presence of Fe2+ and inorg. sulfide. The dithiol protein thioredoxin-A of E. coli acted catalytically in vitro in the presence of thioredoxin reductase and NADPH to promote [2Fe-2S] cluster assembly into apo-SoxR. The regulatory activity of SoxR in vivo, assessed by monitoring the paraquat-mediated induction of a soxS'::lacZ reporter fusion, was significantly lower in a strain lacking both thioredoxin-A and glutathione reductase, which maintains reduced glutaredoxins. Thus, cellular monothiols and dithiol proteins may contribute to SoxR regulation by affecting the disassembly and reassembly of the [2Fe-2S] clusters.

IT 60-23-1, 2-Aminoethanethiol
RL: BAC (Biological activity or effector, except adverse); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(thiol-mediated disassembly and reassembly of [2Fe-2S] clusters in redox-regulated transcription factor SoxR of)

RN 60-23-1 CAPLUS
CN Ethanethiol, 2-amino- (8CI, 9CI) (CA INDEX NAME)

$H_2N-CH_2-CH_2-SH$

REFERENCE COUNT: 43 THERE ARE 43 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

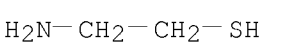
L4 ANSWER 46 OF 88 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1997:278331 CAPLUS
DOCUMENT NUMBER: 126:258875
ORIGINAL REFERENCE NO.: 126:49961a,49964a
TITLE: Cathinone [(R)-(+)-α-aminopropiophenone]: a potent anti-gastric ulcer agent
AUTHOR(S): Al-Gharably, N. M.; Islam, M. W.; Al-Harbi, M. M.; Al-Shabanah, O. A.
CORPORATE SOURCE: Department of Pharmacology, College of Pharmacy, King Saud University, Riyadh, 11451, Saudi Arabia
SOURCE: Research Communications in Alcohol and Substances of Abuse (1996), 17(3&4), 165-184
CODEN: RCAAE3; ISSN: 1080-8388
PUBLISHER: PJD Publications
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Cathinone was studied for its ability to inhibit gastric secretion and to protect the gastric mucosa against the injuries caused by pylorus ligation, nonsteroidal anti-inflammatory drugs (NSAIDs: aspirin, indomethacin, phenylbutazone), reserpine, hypothermic restraint stress, and cysteamine, and as a cytoprotective agent against the effect of necrotizing agents (0.6M HCl, 0.2M NaOH, 80% EtOH, 25% NaCl). It was administered by gastric intubation at 30 and 100 mg/kg to rats fed a standard chow diet. At the doses tested, cathinone produced mucosal protection in the various exptl. models. It provided significant inhibition of gastric mucosal damage induced by pylorus ligation, NSAIDs,

and hypothermic restraint. However, no significant effect was produceed against reserpine-induced ulceration. At 100 mg/kg only, cathinone inhibited the severity and the incidence of duodenal ulceration induced by cysteamine. It also produced a marked cytoprotective effect against all the necrotizing agents used. These results suggest that cathinone possesses both antisecretory and antiulcerogenic effects.

IT 60-23-1, Cysteamine
RL: BAC (Biological activity or effector, except adverse); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(cathinone protection against ulcers induced by)

RN 60-23-1 CAPLUS
CN Ethanethiol, 2-amino- (8CI, 9CI) (CA INDEX NAME)

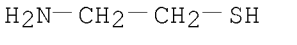


L4 ANSWER 47 OF 88 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1965:18182 CAPLUS
DOCUMENT NUMBER: 62:18182
ORIGINAL REFERENCE NO.: 62:3294a-c
TITLE: An effect of aggregation upon the metabolism of dopamine-1-3H
AUTHOR(S): Welch, Bruce L.; Welch, Ann Marie
CORPORATE SOURCE: Coll. of William & Mary, Williamsburg, VA
SOURCE: Progr. Brain Res. (1964), 8, 201-6
DOCUMENT TYPE: Journal
LANGUAGE: Unavailable

AB After administering dopamine-1-3H to white Swiss mice, radioactivity accumulated in the brains in concns. 60-8-fold greater than could be accounted for by the radioactivity of the blood. After 24 hrs., mice were placed into treatment groups or kept singly. After 7 days, radioactivity remaining in the brains of grouped mice was 2-fold greater than that remaining in the brain of single mice. Radioactivity in 24-hr.-brain exts. completely passed through a chromatographic column which was capable of retaining catechol amines, but only 80% of the radioactivity of grouped mice and 65% of that of single mouse brains passed through without retention, using 7-day-old mouse-brain exts. 30 references.

IT 60-23-1, Ethanethiol, 2-amino-
(effect on O in brain)

RN 60-23-1 CAPLUS
CN Ethanethiol, 2-amino- (8CI, 9CI) (CA INDEX NAME)



L4 ANSWER 48 OF 88 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2004:100662 CAPLUS
DOCUMENT NUMBER: 140:160084
TITLE: Biochips for characterizing biological processes
INVENTOR(S): Kreimer, David I.; Nufert, Thomas H.; Ginzburg, Lev; Yevin, Oleg A.
PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 71 pp., Cont.-in-part of U.S. Ser. No. 925,189.
CODEN: USXXCO
DOCUMENT TYPE: Patent

LANGUAGE: English
FAMILY ACC. NUM. COUNT: 9
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 20040023293	A1	20040205	US 2002-294385	20021114 <--
US 20010053521	A1	20011220	US 2001-815909	20010323 <--
US 20020132371	A1	20020919	US 2001-925189	20010808 <--
WO 2002077558	A2	20021003	WO 2002-US8858	20020322 <--
WO 2002077558	A3	20071122		
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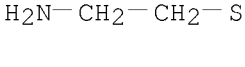
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, AP, EA, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, EP, OA				
AU 2002255883	A1	20021008	AU 2002-255883	20020322 <--
TW 530146	B	20030501	TW 2002-91105672	20020322 <--
US 20030180720	A1	20030925	US 2003-364160	20030211 <--

PRIORITY APPLN. INFO.:		
US 1999-156195P	P	19990927
US 2000-670453	A2	20000926
US 2001-815909	A2	20010323
US 2001-925189	A2	20010808
US 2001-336445P	P	20011114
US 1999-156145P	P	19990927
US 1999-156471P	P	19990927
US 2000-669369	A	20000926
US 2000-669796	A	20000926
US 2001-815828	A	20010323
US 2002-356254P	P	20020211
WO 2002-US8858	W	20020322
US 2002-294385	A2	20021114
US 2002-298725	A2	20021118

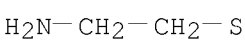
AB This invention includes biochips for anal. of a variety of mols., cell components and cells. Embodiments of this invention include devices and methods for the parallel and/or nearly parallel processing of biol. analytes. Biochips can comprise a substrate, Raman signal-enhancing structures, and receptors selective and/or specific for the analyte(s) to be assayed. Biochips can be read using a Raman reader and can provide for rapid, sensitive, direct assays for physiol. and/or pathophysiol. conditions of interest. Gold-coated quartz slides with silver fractal aggregates as enhancing agents and immobilized reduced glutathione as receptor were used to detect glutathione S-transferase by Raman spectroscopy.

IT 60-23-1, Mercaptoethylamine
RL: DEV (Device component use); NUU (Other use, unclassified); TEM (Technical or engineered material use); USES (Uses)
(as passivation agent; biochips having analyte-specific receptors and enhancing particle structures on substrates for characterizing biol. processes)

RN 60-23-1 CAPLUS
CN Ethanethiol, 2-amino- (8CI, 9CI) (CA INDEX NAME)



L4 ANSWER 49 OF 88 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1989:54101 CAPLUS
DOCUMENT NUMBER: 110:54101
ORIGINAL REFERENCE NO.: 110:8873a,8876a
TITLE: Free radical formation and cell lysis induced by ultrasound in the presence of different rare gases
AUTHOR(S): Kondo, Takashi; Gamson, Janet; Mitchell, James B.; Riesz, Peter
CORPORATE SOURCE: Div. Cancer Treat., Natl. Cancer Inst., Bethesda, MD, 20892, USA
SOURCE: International Journal of Radiation Biology (1988), 54(6), 955-62
CODEN: IJRBE7; ISSN: 0955-3002
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The effect of varying the temperature of cavitation bubbles in aqueous solns. of different rare gases on free radical formation and shearing stress induced by ultrasound was investigated. After sonication with 50-kHz ultrasound, the yield of hydroxyl radicals was measured by spin trapping with 5,5-dimethyl-1-pyrroline N-oxide and the cell lysis of cultured mammalian cells was investigated. The hydroxyl radical yields were in the order Xe > Kr > Ar > Ne > He, in accord with the higher temps. of the cavitation bubbles. However, cell lysis induced by shearing stress was the same for all of the rare gases and independent of their thermal conductivity and the temperature of the cavitation bubbles.
IT 60-23-1, Cysteamine
RL: ANST (Analytical study)
(hydroxyl radical formation and cell lysis response to)
RN 60-23-1 CAPLUS
CN Ethanethiol, 2-amino- (8CI, 9CI) (CA INDEX NAME)



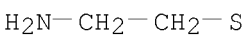
L4 ANSWER 50 OF 88 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1976:55193 CAPLUS
DOCUMENT NUMBER: 84:55193
ORIGINAL REFERENCE NO.: 84:9061a,9064a
TITLE: pH restraints on lettuce fruit germination
AUTHOR(S): Reynolds, T.
CORPORATE SOURCE: Jodrell Lab., R. Bot. Gard., Kew/Richmond/Surrey, UK
SOURCE: Annals of Botany (Oxford, United Kingdom) (1975), 39(162), 797-805
CODEN: ANBOA4; ISSN: 0305-7364
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The effects of buffers with a range of pH values and of concns. low enough to exert negligible osmotic stress on germination of lettuce (Lactuca sativa) seeds were examnd. No restraints were noted except at extremes of pH. Furthermore, inhibition in HCl [7647-01-0] or KOH [1310-58-3] solns. was not evident below concns. of about 0.05M. Acetic acid [64-19-7] or NH4OH [1336-21-6] was very much more inhibitory but their salt, ammonium acetate [631-61-8], only inhibited when its concentration reached a sufficiently high level to operate by osmotic stress. Inhibition by a series of organic acids and bases showed a pos. correlation with the lipophilic nature of the mol., although there were some unexplained exceptions. In contrast with previous cases of germination

inhibition, the effect was not produced by a lowering of the upper temperature cut-off point, but by an overall lowering of total germination at all temps. This indicates a toxic effect of pH extremes rather than a true inhibition.

IT 60-23-1
RL: BIOL (Biological study)
(lettuce seed germination in relation to)

RN 60-23-1 CAPLUS

CN Ethanethiol, 2-amino- (8CI, 9CI) (CA INDEX NAME)



L4 ANSWER 51 OF 88 CAPLUS COPYRIGHT 2008 ACS on STN

ACCESSION NUMBER: 2004:1021429 CAPLUS

DOCUMENT NUMBER: 143:152239

TITLE: On the significant influence of water on the formation mechanism of 5-acetyl-3,4-dihydro-2H-1,4-thiazine

AUTHOR(S): Engel, W.; Schieberle, P.

CORPORATE SOURCE: Deutsche Forschungsanstalt fuer Lebensmittelchemie, Garching bei Muenchen, Germany

SOURCE: Czech Journal of Food Sciences (2004), 22(Spec. Iss.), 120-122
CODEN: CJFSFZ; ISSN: 1212-1800

PUBLISHER: Czech Academy of Agricultural Sciences, Institute of Agricultural and Food Information

DOCUMENT TYPE: Journal

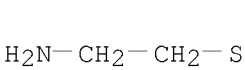
LANGUAGE: English

AB The formation of 5-acetyl-3,4-dihydro-2H-1,4-thiazine in Maillard-type reactions of fructose with cysteamine under dry heating and cooking conditions was studied. Labeling expts. with 2-13C-fructose revealed, that the formation pathways are completely different, depending on the water content of the mixture Under dry heating conditions, 5-(1-13C-acetyl)-3,4-dihydro-2H-1,4-thiazine is formed almost exclusively with the 2-13C of fructose found at the carbonyl carbon of the acetyl group. Under cooking conditions, ADHT is mostly unlabeled and most probably formed from erythrulose. Erythrulose might be generated from 2-13C-fructose by loss of 1-13C-acetic acid, indicated by the high amount of the latter found in the mixture A possible mechanism leading from fructose to erythrulose is postulated.

IT 60-23-1, Cysteamine
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(water effect on erythulose formation from fructose)

RN 60-23-1 CAPLUS

CN Ethanethiol, 2-amino- (8CI, 9CI) (CA INDEX NAME)



REFERENCE COUNT: 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 52 OF 88 CAPLUS COPYRIGHT 2008 ACS on STN

ACCESSION NUMBER: 2002:90253 CAPLUS

DOCUMENT NUMBER: 136:130766

TITLE: Heme oxygenase 1 transcriptional suppressor levels as a diagnostic and prognostic indicator for dementia

INVENTOR(S): Schipper, Hyman M.

PATENT ASSIGNEE(S): The Sir Mortimer B. Davis - Jewish General Hospital, Can.
SOURCE: PCT Int. Appl., 60 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

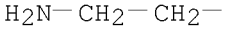
PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002008449	A2	20020131	WO 2001-CA1066	20010725 <--
WO 2002008449	A3	20020906		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
CA 2417134	A1	20020131	CA 2001-2417134	20010725 <--
EP 1303537	A2	20030423	EP 2001-957650	20010725 <--
EP 1303537	B1	20060927		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
JP 2004504366	T	20040212	JP 2002-513931	20010725 <--
AT 340805	T	20061015	AT 2001-957650	20010725
US 20040033563	A1	20040219	US 2003-333880	20030728 <--
US 7105485	B2	20060912		

PRIORITY APPLN. INFO.:			
US 2000-220813P	P	20000725	
WO 2001-CA1066	W	20010725	

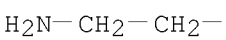
AB The invention relates to an improved method for predicting the onset of, diagnosing, prognosticating and/or treating dementing diseases. The method comprises determining the level of heme oxygenase-1 suppressor (HOS) activity and/or factor in tissue or body fluid obtained from a patient, and comparing said level with the corresponding level of HOS activity and/or factor in corresponding tissue or body fluid obtained from at least one control person. The tissue or body fluid is suitably blood, plasma, lymphocytes, cerebrospinal fluid, urine, saliva, epithelia or fibroblasts. The method is useful where the dementing disease is any of Alzheimer's disease, age-associated cognitive decline, mild cognitive impairment, Parkinson's disease with dementia, progressive supranuclear palsy, vascular (i.e. multi-infarct) dementia, Lewy body dementia, Huntington's disease, Down's syndrome, normal pressure hydrocephalus, corticobasal ganglionic degeneration, multisystem atrophy, head trauma, neurosyphilis, Creutzfeldt-Jacob disease and other prion diseases, HIV and other encephalitides, and metabolic disorders such as hypothyroidism and vitamin B12 deficiency. The method may also prove useful in differentiating the "pseudodementia" of depression from Alzheimer disease. Cysteamine strongly induced heme oxygenase 1 gene expression in cultured astrocytes from rats and control humans, but not from presumptive Alzheimer's disease patients.

IT 60-23-1, Cysteamine
RL: DGN (Diagnostic use); BIOL (Biological study); USES (Uses)
(heme oxygenase induction by, in assays for transcriptional suppressor; heme oxygenase 1 transcriptional suppressor levels as diagnostic and prognostic indicator for dementia)

RN 60-23-1 CAPLUS
CN Ethanethiol, 2-amino- (8CI, 9CI) (CA INDEX NAME)



L4 ANSWER 53 OF 88 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2000:285490 CAPLUS
DOCUMENT NUMBER: 133:70746
TITLE: Noninvasive study of radiation-induced oxidative damage using in vivo electron spin resonance
AUTHOR(S): Miura, Y.; Ozawa, T.
CORPORATE SOURCE: Department of Biochemistry and Isotopes, Tokyo Metropolitan Institute of Gerontology, Tokyo, Japan
SOURCE: Free Radical Biology & Medicine (2000), 28(6), 854-859
CODEN: FRBMEH; ISSN: 0891-5849
PUBLISHER: Elsevier Science Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Nitroxyl radicals injected into a whole body indicate the disappearance of signal intensity of in vivo ESR. The signal decay rates of nitroxyl are influenced by various types of oxidative stress. We examined the effect of X-irradiation on the signal decay rate of nitroxyl in the upper abdomen of mice using in vivo ESR. The signal decay rates increased 1 h after 15 Gy irradiation, and the enhancement was suppressed by preadministration of cysteamine, a radioprotector. These results suggest that the signal decay of nitroxyl in whole mice is enhanced by radiation-induced oxidative damage. The in vivo ESR system probing the signal decay of nitroxyl could provide a noninvasive technique for the study of oxidative stress caused by radiation in a living body.
IT 60-23-1, Cysteamine
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(study of radiation-induced oxidative damage using in vivo ESR: effect of radioprotectant)
RN 60-23-1 CAPLUS
CN Ethanethiol, 2-amino- (8CI, 9CI) (CA INDEX NAME)



REFERENCE COUNT: 29 THERE ARE 29 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

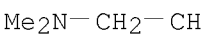
L4 ANSWER 54 OF 88 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1992:405040 CAPLUS
DOCUMENT NUMBER: 117:5040
ORIGINAL REFERENCE NO.: 117:1043a,1046a
TITLE: Immobilized amines and basic amino acids as mimetic heparin-binding domains for cell surface proteoglycan-mediated adhesion
AUTHOR(S): Massia, Stephen P.; Hubbell, Jeffrey A.
CORPORATE SOURCE: Dep. Chem. Eng., Univ. Texas, Austin, TX, 78712-1062, USA
SOURCE: Journal of Biological Chemistry (1992), 267(14), 10133-41
CODEN: JBCHA3; ISSN: 0021-9258
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Diamines covalently coupled to glass substrates promoted human foreskin fibroblast adhesion in the absence of serum. These diamine-derivatized substrates were produced by coupling ethylene diamine, N-methylaminoethylamine, and N,N-dimethylaminoethylamine (NNDMAEA), to sulfonyl chloride-activated glass. Electron spectroscopy for chemical anal. demonstrated that the diamines were coupled via their primary amine ends to produce a surface-bound secondary amine linked to a free amino moiety via a 2-carbon spacer. NNDMAEA-modified substrates containing free tertiary amines supported the highest degree of cell spreading (73% actively spreading cells) and the most extensive cytoskeletal organization. Both the free tertiary and surface-bound secondary amines were required for cell spreading. Lysine- and arginine-grafted substrates supported cell spreading and cytoskeletal organization similar to that on NNDMAEA-modified substrates. Although some stress fibers were observed within spread cells on these substrates, focal contacts did not form. Heparinase treatment did not inhibit cell attachment or spreading to the diamine-derivatized substrates, however chondroitinase ABC inhibited cell attachment and spreading on all substrates; heparinase inhibited spreading on lysine- and arginine-derivatized substrates to a lesser extent. These results imply that cell attachment to these substrates was mediated primarily by cell surface chondroitin sulfate proteoglycans. This study demonstrates that covalently grafted NNDMAEA, lysine, and arginine can mimic the adhesion-promoting activity of the glycosaminoglycan-binding domains of cell adhesion proteins. This study also demonstrates that the interaction with these proteoglycans depends in a very sensitive manner on the particular structure of the immobilized amine.

IT 108-02-1D, immobilized
RL: BIOL (Biological study)
(on glass, human fibroblast adhesion to, structure in relation to)

RN 108-02-1 CAPLUS

CN Ethanethiol, 2-(dimethylamino)- (CA INDEX NAME)



L4 ANSWER 55 OF 88 CAPLUS COPYRIGHT 2008 ACS on STN

ACCESSION NUMBER: 1989:192268 CAPLUS

DOCUMENT NUMBER: 110:192268

ORIGINAL REFERENCE NO.: 110:31901a,31904a

TITLE: Sulfur-containing fatty acid amide derivatives as peptic ulcer inhibitors

INVENTOR(S): Iwai, Masakazu; Arakawa, Yoshio; Fukaya, Tsutomu; Yokoyama, Kazumasa

PATENT ASSIGNEE(S): Green Cross Corp., Japan

SOURCE: Jpn. Kokai Tokkyo Koho, 4 pp.
CODEN: JKXXAF

DOCUMENT TYPE: Patent

LANGUAGE: Japanese

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
JP 63301857	A	19881208	JP 1987-72731	19870325 <--

PRIORITY APPLN. INFO.: JP 1987-7246 A1 19870113

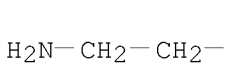
AB Amide derivs. prepared from S-containing amines and C4-unsatd. fatty acids are useful as peptic ulcer inhibitors. A solution of 2.28 g cystamine in CHCl3 was treated with 3.24 g crotonoyl chloride in the presence of Et3N at room

temperature for 1.5 h to give 2.32 g N,N'-dicrotonoylcystamine, 500 mg of which was treated with Bu3P in aqueous MeOH at room temperature for 1 h, then 357 mg ClCH2CONH2 was added and the mixture was stirred for 1 h at room temperature to give 353 mg S-carbamoylmethyl-N-crotonoylcysteamine (I). I, at 20 mg/kg, inhibited stress-induced ulcer in rats by 78.3%.

IT 60-23-1DP, Cysteamine, alkylated, fatty acid amide derivs.
RL: PREP (Preparation)
(preparation of, as peptic ulcer inhibitors)

RN 60-23-1 CAPLUS

CN Ethanethiol, 2-amino- (8CI, 9CI) (CA INDEX NAME)



L4 ANSWER 56 OF 88 CAPLUS COPYRIGHT 2008 ACS on STN

ACCESSION NUMBER: 2001:825250 CAPLUS

DOCUMENT NUMBER: 136:354042

TITLE: Oxidative metabolism in HIV-infected macrophages: Role of glutathione and pharmacological approach

AUTHOR(S): Mialocq, P.; Oiry, J.; Puy, J. Y.; Rimaniol, A. C.; Imbach, J. L.; Dormont, D.; Clayette, P.

CORPORATE SOURCE: CEA, service de neurovirologie, DSV/DRM, CRSSA, EPHE, IPSC, Fontenay-aux-Roses, 92265, Fr.

SOURCE: Pathologie Biologie (2001), 49(7), 567-571
CODEN: PTBIAN; ISSN: 0031-3009

PUBLISHER: Editions Scientifiques et Medicales Elsevier

DOCUMENT TYPE: Journal

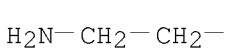
LANGUAGE: English

AB Oxidative stress and glutathione deficiency seem to play a major role in the pathogenesis of HIV infection, as suggested by the increased survival of HIV-infected patients treated with N-acetylcysteine, a prodrug of glutathione. However, beneficial effects of GSH-replenishing drugs are restricted in vivo by the high concns. needed to obtain biol. effects and their low bioavailability. In this study, we evaluated the antiretroviral and antioxidant activities of new more lipophilic GSH-replenishing mols., in macrophages infected in vitro with HIV-1. In these exptl. conditions, a prodrug of N-acetylcysteine and β-mercaptoethylamine, I-152 demonstrated a potent anti-HIV activity, increased intracellular GSH level, and decreased TNF-α production Altogether, these results suggest that I-152 could be beneficial as adjuvant therapy of antiretrovirals in HIV-infected patients, especially in those with damages to the central nervous system or with mitochondrial damages associated with highly active antiretroviral therapy.

IT 60-23-1, β-Mercaptoethylamine
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(pharmaceutically induced glutathione production and oxidative metabolism in HIV-infected macrophages)

RN 60-23-1 CAPLUS

CN Ethanethiol, 2-amino- (8CI, 9CI) (CA INDEX NAME)



REFERENCE COUNT: 21 THERE ARE 21 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 57 OF 88 CAPLUS COPYRIGHT 2008 ACS on STN

ACCESSION NUMBER: 1995:905068 CAPLUS

DOCUMENT NUMBER: 124:6277
ORIGINAL REFERENCE NO.: 124:1355a,1358a
TITLE: Redox perturbations in cysteamine-stressed astroglia: implications for inclusion formation and gliosis in the aging brain
AUTHOR(S): Manganaro, Fortunato; Chopra, Vikramjit S.; Mydlarski, Marc B.; Bernatchez, Gerald; Schipper, Hyman M.
CORPORATE SOURCE: Lady Davis Institute Medical Research, Sir Mortimer B. Davis-Jewish General Hospital, Peop. Rep. China
SOURCE: Free Radical Biology & Medicine (1995), 19(6), 823-35
CODEN: FRBMEH; ISSN: 0891-5849
PUBLISHER: Elsevier
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The aminothiol compound, cysteamine (CSH), induces astrocyte hypertrophy (gliosis) and the appearance of autofluorescent, peroxidase-pos. cytoplasmic granules in these cells akin to changes that occur spontaneously in astroglia of the aging periventricular brain. Paradoxically, CSH damages astroglial mitochondria (granule precursors) while protecting these cells from subsequent H2O2 and mechanoenzymic stress. In this study, in vitro CSH administration significantly increased manganese superoxide dismutase (MnSOD) activity in cultured astroglia. Immunoblot and Northern analyses indicated that MnSOD protein and mRNA levels were increased in cultured astrocytes after 3-6 days of CSH treatment. Systemic administration of CSH also significantly augmented MnSOD activity in the intact diencephalon. CSH caused a pronounced (6-fold), but transient, increase in the level of reduced glutathione (GSH) in cultured astrocytes. In contrast, catalase and glutathione reductase (GR) activities were suppressed, whereas copper-zinc superoxide dismutase (CuZnSOD) activity remained unchanged both in cultured astroglia and in the intact diencephalon following CSH treatment. Glutathione peroxidase (GP) activity was increased after 3 and 48 h of CSH treatment and then declined below control levels in cultured astrocytes. CSH inhibited the formation of thiobarbituric acid-reactive products (TBAR) in whole astrocyte monolayers, although it promoted TBAR formation in suspensions of isolated astroglial mitochondria. CSH-related oxidative stress may accelerate aging-related changes in astroglial mitochondria while conferring cytoprotection to these cells by stimulating the upregulation of various heat shock proteins and MnSOD. These cytoprotective responses may facilitate astrocyte survival and the development of reactive gliosis in the face of concomitant neuronal degeneration. CSH-treated astrocytes may serve as a model for the (dys)regulation of neuroglial MnSOD and other antioxidant enzymes in the aging and degenerating nervous system.

IT 60-23-1, Cysteamine
RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)
(redox perturbations in cysteamine-stressed astroglia and inclusion formation and gliosis in the aging brain)
RN 60-23-1 CAPLUS
CN Ethanethiol, 2-amino- (8CI, 9CI) (CA INDEX NAME)

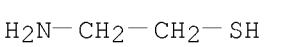
$H_2N-CH_2-CH_2-SH$

L4 ANSWER 58 OF 88 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1995:880472 CAPLUS
DOCUMENT NUMBER: 123:306532
ORIGINAL REFERENCE NO.: 123:54663a,54666a

TITLE: Inhibition of apoptosis by antioxidants in the human HL-60 leukemia cell line
AUTHOR(S): Verhaegen, Steven; McGowan, Adrian J.; Brophy, Alan R.; Fernandes, Richard S.; Cotter, Thomas G.
CORPORATE SOURCE: Tumor Biology Lab., Univ. College Cork, Co., Cork, Ire.
SOURCE: Biochemical Pharmacology (1995), 50(7), 1021-9
CODEN: BCPCA6; ISSN: 0006-2952
PUBLISHER: Elsevier
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Cell death via apoptosis is an important event involved in a number of immunol. processes. Recently, apoptosis has been associated with oxidative stress in a number of cell systems. Here the authors assessed the inhibitory capacity of different antioxidants on UV- and drug-induced apoptosis in the human leukemic cell line, HL-60. The authors found that the oxygen radical scavenger butylated hydroxyanisole (BHA), the radioprotector cysteamine and the metal chelators pyrrolidinedithiocarbamate (PDTC), diethyldithiocarbamate (DEDTC), and dimethyldithiocarbamate (DMDTC), were able to significantly inhibit nuclear fragmentation and reduce the formation of apoptotic bodies in UV-irradiated human leukemic cells. Both BHA and PDTC were found to reduce DNA fragmentation as assessed by in situ DNA nick-end labeling and quantification thereof using fluorescence flow cytometry. In addition to inhibiting UV-induced apoptosis, PDTC was also capable of reducing the amount of apoptosis induced by a range of cytotoxic drugs, such as actinomycin D, camptothecin, etoposide, and melphalan, whereas BHA and cysteamine were not as effective in these cases after more than four hours in culture when compared to PDTC. To further elucidate the working mechanism of PDTC, the authors have looked at the effect of PDTC on DNA fragmentation in isolated nuclei, under conditions that promote activation of endogenous endonuclease involved in a apoptosis. In contrast to ZnCl2, a potent inhibitor of endonuclease activity, PDTC was unable to inhibit DNA-ladder formation in this assay. Taken together, these results indicate that oxygen radicals may have a central role to play in the induction of apoptosis and that dithiocarbamates can serve as potent inhibitors of apoptosis induced by a wide variety of stimuli.

IT 60-23-1, Cysteamine
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(inhibition of apoptosis induced by cytotoxic drugs and UV irradiation by antioxidants in human HL-60 leukemia cell line in relation to oxygen radicals)
RN 60-23-1 CAPLUS
CN Ethanethiol, 2-amino- (8CI, 9CI) (CA INDEX NAME)



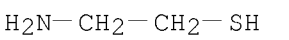
L4 ANSWER 59 OF 88 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1987:193951 CAPLUS
DOCUMENT NUMBER: 106:193951
ORIGINAL REFERENCE NO.: 106:31417a,31420a
TITLE: Biochemical changes in tissue catecholamines and serotonin in duodenal ulceration caused by cysteamine or propionitrile in the rat
AUTHOR(S): Szabo, S.; Horner, H. C.; Maull, H.; Schnoor, J.; Chiueh, C. C.; Palkovits, M.

CORPORATE SOURCE: Dep. Pathol., Brigham Women's Hosp., Boston, MA, 02115, USA
SOURCE: Journal of Pharmacology and Experimental Therapeutics (1987), 240(3), 871-8
CODEN: JPETAB; ISSN: 0022-3565
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Previous structure-activity and pharmacol. studies with duodenal ulcerogens cysteamine and propionitrile implicating catecholamines in the pathogenesis of duodenal ulceration were followed up by dose- and time-response biochem. investigations to assess the importance of monoamines in the development of duodenal ulcers. The duodenal ulcerogens caused a dose- and time-dependent depletion of norepinephrine in virtually all the tissues examined The effect was maximal 4 or 7 h after cysteamine or propionitrile, and norepinephrine levels returned to normal in 24 h. Dopamine changes were selective and often biphasic, e.g., elevation in adrenals, biphasic in brain cortex, hippocampus and midbrain, but uniformly decreasing in glandular stomach and duodenum. In the median eminence, dopamine levels decreased by 181 and 324% at 15 and 30 min, resp., after cysteamine, but neither dopamine nor 3,4-dihydroxyphenylacetic acid was modified in the periventricular nucleus. Serotonin levels were relatively stable, revealing slight elevations or no changes in most of the tissues. The turnover of norepinephrine was accelerated by both chems. in virtually all brain regions, but dopamine turnover was affected only in few areas e.g., in the corpus striatum and medulla oblongata cysteamine decreased dopamine turnover, whereas propionitrile first (at 1 h) accelerated then (at 8 h) suppressed it. Correlation of ulcer intensity after a single dose of cysteamine with concns. of monoamines revealed a neg. association between dopamine levels in the brain and duodenal ulcer severity. Thus, the development of exptl. duodenal ulcers is preceded and accompanied by change in central and peripheral tissue levels of catecholamines. Inasmuch as nonspecific stress, which alone is unable to cause duodenal ulcer, is accompanied by tissue norepinephrine depletion, the unusual changes in dopamine levels might have a pathogenic role in duodenal ulceration and represent a pharmacol. target for preventive therapeutic intervention.

IT 60-23-1, Cysteamine
RL: BIOL (Biological study)
(duodenal ulcers from, catecholamines of tissues in)

RN 60-23-1 CAPLUS
CN Ethanethiol, 2-amino- (8CI, 9CI) (CA INDEX NAME)



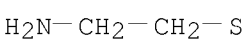
L4 ANSWER 60 OF 88 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1966:440495 CAPLUS
DOCUMENT NUMBER: 65:40495
ORIGINAL REFERENCE NO.: 65:7598b-e
TITLE: Chemical protection against ionizing radiations in mammals
AUTHOR(S): Bacq, Z. M.
CORPORATE SOURCE: Univ. Liege
SOURCE: Bulletin de l'Academie Royale de Medecine de Belgique (1966), 6(2), 115-41
CODEN: BARMAW; ISSN: 0001-4168
DOCUMENT TYPE: Journal
LANGUAGE: French

AB The reasons for the radioprotective effect of S compds. on mammals are not clear. The classical hypothesis of hypoxia, mixed disulfides, and free radicals scavenging are inadequate to explain the protection given to rats or mice by cysteamine or cystamine injections. Intermediate reactions are explained. New concepts of biochem. shock are defined; fixing of the protective substance to the proteins or other macromols of the intracellular structures (mainly mitochondria) changes the permeability of these structures provokes the liberation of certain substances, and inhibits the utilization of carbohydrates. In the ensuing hrs. the cell restores the normal equilibrium slowly. Two to 3 min. after intraperitoneal injection of a radioprotective dose of cysteamine or cystamine to rats or mice, radiation induces mitochondrial lesions in the radiosensitire organs (spleen, thymus, duodenal mucosa), defective carbohydrate metabolism, and a drop in O consumption and in R.Q. Some of these can also be observed in isolated systems (liver homogenates, isolated mitochondria). For the development of maximum protection against irradiation, a time lapse of 10 min. is necessary after injection of cysteamine to mice, while cystamine gives a high degree of protection as early as 2 min. after injection. The degree of protection falls at 6 min. and again rises at 10 min. postinjection. Afterwards, it decreases in a way very similar to the protection afforded by cysteamine. Biochem. shock is avoided by feeding 1% cystamine with food. Continuous administration of this does not protect mice against continuous exposure to ^{137}Cs γ-rays at low dosages (from 1 to 0.026 r./min.). Mice fed cystamine with food but not irradiated also lose weight This is not due to toxicity but could be attributed to lessened consumption of the food. Various expts. are described to stress the hypothesis of biochem. shock, and are followed by a discussion.

IT 60-23-1, Ethanethiol, 2-amino-
(in radiation-damage prevention)

RN 60-23-1 CAPLUS

CN Ethanethiol, 2-amino- (8CI, 9CI) (CA INDEX NAME)



L4 ANSWER 61 OF 88 CAPLUS COPYRIGHT 2008 ACS on STN

ACCESSION NUMBER: 1964:48300 CAPLUS

DOCUMENT NUMBER: 60:48300

ORIGINAL REFERENCE NO.: 60:8524d-f

TITLE: Proliferation of mast cells in the bone marrow of rats after feeding β-aminopropionitrile (BAPN) and β-mercaptoethylamine (BMEA)

AUTHOR(S): Takeoka, Osamu; Angevine, D. Murray; Lalich, Joseph J.

CORPORATE SOURCE: Univ. of Wisconsin Med. School, Madison

SOURCE: American Journal of Pathology (1963), 43(4), 639-50
CODEN: AJPAA4; ISSN: 0002-9440

DOCUMENT TYPE: Journal

LANGUAGE: Unavailable

AB Feeding of BAPN and BMEA to rats increased the number of mast cells in the diaphyseal marrow of long bones and spinous processes of the vertebrae. However, they induced no increase in the number of mast cells in the marrow of the epiphyseal ends in long bones and only min. mast cell stimulation in the vertebral bodies. Mast cells were not observed in the peripheral blood even when there was a conspicuous increase in their nos. in bone marrow. The number of mast cells in the lymph nodes remained within normal limits following BAPN and BMEA feeding. It is suggested that the increase in mast cell nos. in bone marrow may be related to the mech.

stress produced by muscle tension. Mech. stress tends to be greater on long and thin bones. It would, therefore, seem reasonable to assume that when bones were weakened by reduction in density as a result of BAPN or BMEA intoxication, they became more susceptible to distortion so that the effects of mech. stress would be greater. Under such conditions, soft tissue such as marrow contained within the bones would probably be subjected to continuous distortion.

IT 60-23-1, Ethanethiol, 2-amino-
(effect on mast cell proliferation in bone marrow)

RN 60-23-1 CAPLUS

CN Ethanethiol, 2-amino- (8CI, 9CI) (CA INDEX NAME)

L4 ANSWER 62 OF 88 CAPLUS COPYRIGHT 2008 ACS on STN

ACCESSION NUMBER: 2003:89879 CAPLUS

DOCUMENT NUMBER: 138:271964

TITLE: Thiyl Radicals Abstract Hydrogen Atoms from the αC-H Bonds in Model Peptides: Absolute Rate Constants and Effect of Amino Acid Structure

AUTHOR(S): Nauser, Thomas; Schoeneich, Christian

CORPORATE SOURCE: Department of Pharmaceutical Chemistry, University of Kansas, Lawrence, KS, 66047, USA

SOURCE: Journal of the American Chemical Society (2003), 125(8), 2042-2043
CODEN: JACSAT; ISSN: 0002-7863

PUBLISHER: American Chemical Society

DOCUMENT TYPE: Journal

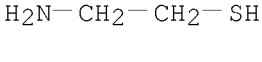
LANGUAGE: English

AB Thiyl radicals are important intermediates in biol. oxidative stress and enzymic reactions. On the basis of the homolytic bond dissociation energies (BDEs) only, the αC-H bonds of peptides and proteins would present suitable targets for hydrogen abstraction by thiyl radicals. However, addnl. parameters such as polar and conformational effects may control such hydrogen-transfer processes. To evaluate the potential of thiyl radicals for hydrogen abstraction from αC-H bonds, the authors provide the first absolute rate consts. for these reactions with model peptides. Thiyl radicals react with αC-H bonds with rate consts. between 1.7 + 103 M-1 s-1 (N-acetylproline amide) and 4 + 105 M-1 s-1 (sarcosine cyclic dipeptide). However, the correlation of rate consts. with BDEs is poor. Rather, these reactions may be controlled by conformation and dynamic flexibility around the αC-H bonds.

IT 60-23-1, Cysteamine
RL: CPS (Chemical process); PEP (Physical, engineering or chemical process); RCT (Reactant); PROC (Process); RACT (Reactant or reagent)
(absolute rate consts. and effect of amino acid structure on abstraction of hydrogen atoms from model peptides by thiyl radicals)

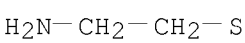
RN 60-23-1 CAPLUS

CN Ethanethiol, 2-amino- (8CI, 9CI) (CA INDEX NAME)



REFERENCE COUNT: 22 THERE ARE 22 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 63 OF 88 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1991:226922 CAPLUS
DOCUMENT NUMBER: 114:226922
ORIGINAL REFERENCE NO.: 114:38225a,38228a
TITLE: Involvement of sulfhydryls in the protective mechanism of gastric mucosa
AUTHOR(S): Li, Tie; Zhang, Xijin
CORPORATE SOURCE: Dep. Physiol., Beijing Med. Univ., Beijing, Peop. Rep. China
SOURCE: Shengli Xuebao (1990), 42(6), 571-7
CODEN: SLHPAH; ISSN: 0371-0874
DOCUMENT TYPE: Journal
LANGUAGE: Chinese
AB The role was studied of nonprotein sulfhydryl (NPSH) in the protective mechanism of gastric mucosa. During the development of gastric injury by acidified ethanol (AE) gavage or restraint-cold stress (RCS), NPSH content in gastric mucosa decreased. Pretreatment with cysteamine (cys) or GSH could prevent gastric mucosa from injury induced by AE. The activity of glutathione reductase in gastric mucosa was inhibited consistently in the time course with NPSH decrease after AE gavage or RCS. Malondialdehyde (MDA) level in the mucosa increased after AE gavage and DMSO, a free radical scavenger, could reduce AE induced injury. The above results suggest that NPSH in gastric mucosa might be involved in the local protective mechanism through its free radical scavenging activity, and the decrease of NPSH in gastric mucosa resulted from the inhibition of glutathione reductase activity and the increase of free radical production may be an important step in the development of injury.
IT 60-23-1, Cysteamine
RL: BIOL (Biological study)
(stomach mucosa injury prevention by)
RN 60-23-1 CAPLUS
CN Ethanethiol, 2-amino- (8CI, 9CI) (CA INDEX NAME)



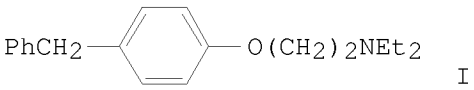
L4 ANSWER 64 OF 88 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1991:441 CAPLUS
DOCUMENT NUMBER: 114:441
ORIGINAL REFERENCE NO.: 114:83a,86a
TITLE: Examination of the potential antiulcer activity of the calcium antagonist propyl-methylenedioxyindene. III. Lack of effect on cysteamine-induced duodenal ulcers in rats
AUTHOR(S): Wong, Wai Shiu Fred; Rahwan, Ralf G.; Stephens, Robert L., Jr.
CORPORATE SOURCE: Coll. Med., Ohio State Univ., Columbus, OH, 43210, USA
SOURCE: Pharmacology (1990), 41(4), 215-23
CODEN: PHMGBN; ISSN: 0031-7012
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Since propyl-methylenedioxyindene (pr-MDI) exhibits significant protective effects against stress-induced ulcers in rats at subcardiovascular doses (10-30 mg/kg, i.p.), the aim of the present study was to explore the effect of this intracellular calcium antagonist on cysteamine-induced duodenal ulcers at the same low doses. Duodenal ulcers were induced in rats with a single dose of cysteamine (425 mg/kg, s.c.), which produced an 80% ulcer incidence within 24 h without affecting gastric acid concentration Administration of pr-MDI (10 and 30 mg/kg, i.p.) at

0, 6 and 12 h post-cysteamine did not afford protection against ulceration. On the other hand, atropine (10 mg/kg, s.c., administered at 0, 6 and 12 h post-cysteamine) resulted in a 69% inhibition of ulceration, and the antacid Maalox (2 mL, administered p.o. at 0, 2, 4, 6 and 12 h post-cysteamine) completely prevented ulceration. The failure of pr-MDI to protect against duodenal ulceration is discussed in relation to its pharmacol. mechanism of action and the pathogenetic mechanism of action of cysteamine.

IT 60-23-1, Cysteamine
RL: BIOL (Biological study)
(duodenal ulcer induction by, propylmethylenedioxyindene effect on, as calcium antagonist, pathogenesis in relation to)

RN 60-23-1 CAPLUS
CN Ethanethiol, 2-amino- (8CI, 9CI) (CA INDEX NAME)

L4 ANSWER 65 OF 88 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1989:33566 CAPLUS
DOCUMENT NUMBER: 110:33566
ORIGINAL REFERENCE NO.: 110:5481a,5484a
TITLE: A novel non-H1, non-H2 histamine antagonist protects against cysteamine-induced duodenal ulcers in rats
AUTHOR(S): Glavin, Gary B.; Brandes, Lorne J.
CORPORATE SOURCE: Fac. Med., Univ. Manitoba, Winnipeg, MB, R3E 0W3, Can.
SOURCE: Pharmacology (1988), 37(5), 277-80
CODEN: PHMGBN; ISSN: 0031-7012
DOCUMENT TYPE: Journal
LANGUAGE: English
GI

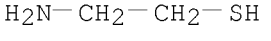


I

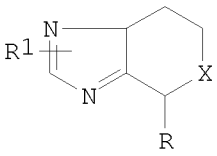
AB A newly synthesized p-diphenylmethane derivative, N,N-diethyl-2-[4-(phenylmethyl)phenoxy]ethanamine (DPPE, I) binds with high affinity to the microsomal anti-estrogen binding site (AEBS). Recent data suggest that the DPPE/AEBS binding site is closely related to a novel low-affinity, non-H1, non-H2 histamine site which may be associated with a Ca+ channel. It was previously shown that DPPE markedly reduces stress-induced and EtOH-induced gastric ulcers and attenuates gastric acid secretion. DPPE also profoundly reduces cysteamine-induced duodenal ulcers in rats.

IT 60-23-1, Cysteamine
RL: BIOL (Biological study)
(duodenal ulcer induction by, diphenylmethane derivative protection against)

RN 60-23-1 CAPLUS
CN Ethanethiol, 2-amino- (8CI, 9CI) (CA INDEX NAME)



L4 ANSWER 66 OF 88 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1986:102273 CAPLUS
DOCUMENT NUMBER: 104:102273
ORIGINAL REFERENCE NO.: 104:16031a,16034a
TITLE: Bicyclic compounds with potential antiulcer and/or antisecretory activity. II. 1(or 3),4,6,7-Tetrahydro-1(3)H-pyrano[3,4-d]imidazoles and 1(or 3),4,6,7-tetrahydro-1(3)H-thiopyrano[3,4-d]imidazoles
AUTHOR(S): Scarponi, U.; Cimaschi, R.; Arcari, G.; Toti, D.; Ballabio, M.; Gandini, E.; De Castiglione, R.
CORPORATE SOURCE: Farmitalia Carlo Erba S.p.A., Milan, Italy
SOURCE: Farmaco, Edizione Scientifica (1986), 41(1), 23-40
CODEN: FRPSAX; ISSN: 0430-0920
DOCUMENT TYPE: Journal
LANGUAGE: English
GI



I, R=YNHC(Z)NHR^2

II, R=CH$_2$NR^{3}R^4

AB Twenty-three title compds. [I; R1 = H or 1-Et; R2 = Me or iso-Pr; X = O or S; Y = CH2 or CH2S(CH2)2; Z = O, S, NCN, or CHNH2; II; R1 = H or 1- or 3-Et; R3 = H, Me, CH2Ph, 4-chlorobenzyl, or methylenedioxyphenylmethylene; R4 = H, iso-Pr, cyclopentyl, cyclohexyl, 4-chlorobenzyl, etc.; R3R4 = (CH2)5 or (CH2)2O(CH2)2; X = O or S] were prepared in several steps starting with the cyclization of 4-(2-hydroxyethyl)imidazole [872-82-2] or 4-(2-mercaptoethyl)imidazole [20979-12-8] with aminoacetaldehyde diethylacetal [645-36-3]. I and II were tested for antiulcer activity in a stress-induced ulcer model in rats, H2-receptor antagonism in isolated guinea pig atria, and anticholinergic activity and toxicity in mice. Compds. exhibiting substantial antiulcer potency at the screening dose (5 mg/kg) were evaluated by full dose-range studies. None of the compds. showed anticholinergic, antihistaminic, or significant gastric antisecretory action (tested for only a few compds.). Some structure-activity relations are considered.
IT 60-23-1
RL: RCT (Reactant); RACT (Reactant or reagent)
(reaction of, with chloromethyltetrahydropyranoimidazole hydrochloride)
RN 60-23-1 CAPLUS
CN Ethanethiol, 2-amino- (8CI, 9CI) (CA INDEX NAME)

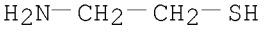
$H_2N-CH_2-CH_2-SH$

L4 ANSWER 67 OF 88 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1984:435232 CAPLUS
DOCUMENT NUMBER: 101:35232

ORIGINAL REFERENCE NO.: 101:5469a,5472a
TITLE: Factors associated with the preincubation effect of hypoxic cell sensitizers in vitro and their possible implications in chemosensitization
AUTHOR(S): Roizin-Towle, Laurie; Biaglow, John E.; Meltzer, Herbert L.; Varnes, Marie E.
CORPORATE SOURCE: Coll. Physicians Surg., Columbia Univ., New York, NY, 10032, USA
SOURCE: Radiation Research (1984), 98(3), 506-18
CODEN: RAREAE; ISSN: 0033-7587
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The enhancement of melphalan toxicity was observed by preincubation of V-79-379A cells in spinner culture with multiple doses of misonidazole (I) or SR 2508 under hypoxic conditions. Chemosensitization was a function of sensitizer concentration and duration of exposure to the alkylating agent. A preincubation exposure of cells with 5 mM I reduced endogenous cell thiols to <5% of controls and enhanced melphalan toxicity by 4.7-fold. Cells preincubated with I not only had lower levels of nonprotein thiols, but also had altered levels of intracellular Ca and a lower threshold to oxidative stress as measured by toxicity to cysteamine or H2O2. Preincubated cells, hypoxic cells, and cells receiving moderate hyperthermia (42.5° for 3 h) all showed increased sensitivity to either cysteamine or H2O2. The increased killing of preincubated cells by cysteamine was similar to that of H2O2, and the reduction of cysteamine toxicity by catalase indicated that H2O2 was the major reaction associated with this effect. Thus preincubated cells exhibit a variety of biol. effects that may influence their response to further treatment with drugs or radiation, especially where peroxidative and free radical mechanisms are involved. The depletion of endogenous thiols, Ca disturbance, and vulnerability to oxidative stress are factors to be considered when interpreting mechanisms of combined drug action and effects that may potentially be exploited in terms of therapeutic gains.

IT 60-23-1
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(V-79 cells sensitivity to)
RN 60-23-1 CAPLUS
CN Ethanethiol, 2-amino- (8CI, 9CI) (CA INDEX NAME)



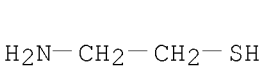
L4 ANSWER 68 OF 88 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1983:515830 CAPLUS
DOCUMENT NUMBER: 99:115830
ORIGINAL REFERENCE NO.: 99:17671a,17674a
TITLE: On the cytoprotective action of sulfhydryl-containing substances
AUTHOR(S): Balint, Gabor A.; Varro, Vince
CORPORATE SOURCE: 1st Dep. Med., Univ. Med. Sch., Szeged, H-6701, Hung.
SOURCE: Acta Physiologica Academiae Scientiarum Hungaricae (1982), 60(3), 139-42
CODEN: APACAB; ISSN: 0001-6756
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The sulfhydryl substances cysteine [52-90-4] (100 mg/kg, orally), glutathione [70-18-8] (100 mg/kg, orally), dicaptol [59-52-9] (10 mg/kg, i.p.), and cysteamine [60-23-1] (100 mg/kg, orally) protected

rats against indomethacin-induced gastric ulcer but potentiated the ulcerogenic effect of immobilization stress. Thus, these sulfhydryl-containing compds. are not cytoprotective in all kinds of exptl. ulcer models.

IT 60-23-1
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(ulcer response to, cytoprotective action in relation to)

RN 60-23-1 CAPLUS
CN Ethanethiol, 2-amino- (8CI, 9CI) (CA INDEX NAME)

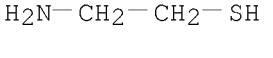


L4 ANSWER 69 OF 88 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1980:530113 CAPLUS
DOCUMENT NUMBER: 93:130113
ORIGINAL REFERENCE NO.: 93:20740h,20741a
TITLE: Experimental studies on the pathogenesis of peptic ulcer. On duodenal ulcer caused by cysteamine administration
AUTHOR(S): Tsunoda, Satoru; Yabana, Tsuyoshi
CORPORATE SOURCE: Dep. Intern. Med., Sapporo Med. Coll., Sapporo, Japan
SOURCE: Sapporo Igaku Zasshi (1980), 49(3), 281-302
CODEN: SIZSAR; ISSN: 0036-472X
DOCUMENT TYPE: Journal
LANGUAGE: Japanese

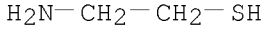
AB Atropine, cimetidine and somatostatin inhibited duodenal ulcer formation and gastric acid and pepsin secretions in cysteamine-treated rats. These agents did not always exert an influence on the increased values of blood gastrin and corticosterone levels arising from cysteamine administration. Degeneration and desquamation of the epithelial columnar cells, constituting duodenal villi, occurred after cysteamine administration. These mucosal lesions were significantly decreased by pretreatment with these gastric inhibitory agents. Acute gastric mucosal lesions induced by cold restraint stress and oral administration of indomethacin were inhibited by cysteamine administration. These phenomena seemed to participate in the increase of neutral mucoprotein and mucin-like glycoprotein M1 fraction induced by cysteamine administration. Duodenal mucosal lesions induced by cysteamine administration seemed to be prevented to some degree in rats kept under cold restraint stress and the prevention mechanism appeared to be related to the increase of Brunner's gland secretion. Local mucosal lesions following the intraduodenal infusion of HCl and pepsin solns. were similar to those produced by cysteamine administration. Abnormalities of the gastric mucosal barrier appeared to be involved in gastric ulcer formation. Excess secretion of gastric acid and pepsin, disturbance of blood circulation in the duodenal mucosa, and marked changes of several humoral factors participate in the formation and development of duodenal ulcers.

IT 60-23-1
RL: BIOL (Biological study)
(duodenum ulceration by, pathogenesis of)

RN 60-23-1 CAPLUS
CN Ethanethiol, 2-amino- (8CI, 9CI) (CA INDEX NAME)

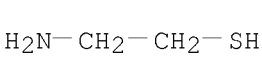


L4 ANSWER 70 OF 88 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1968:27385 CAPLUS
DOCUMENT NUMBER: 68:27385
ORIGINAL REFERENCE NO.: 68:5275a,5278a
TITLE: Radiation effects and adrenal cortex. III. Inhibition of corticosteroid increased by cysteamine after whole-body irradiation
AUTHOR(S): Flemming, Kurt; Geierhaas, Bruno
CORPORATE SOURCE: Univ. Freiburg/Br., Freiburg/Br., Fed. Rep. Ger.
SOURCE: International Journal of Radiation Biology and Related Studies in Physics, Chemistry and Medicine (1967), 13(1), 13-19
CODEN: IJRBA3; ISSN: 0020-7616
DOCUMENT TYPE: Journal
LANGUAGE: English
AB When rats were injected i.p. with cysteamine (100 mg./kg.) 5 min. before whole-body x-irradiation, the previously reported (loc. cit.) biphasic increase of the corticosteroid levels in the adrenal glands (the 1st increase occurred 2.5 hrs. and the 2nd increase occurred 72 hrs. after irradiation) and the blood was significantly inhibited. This radioprotective effect was basically the same after midlethal (750 r.) and totally lethal (1000 r.) radiation doses. Both the first and second corticosteroid reaction are specific effects of irradiation The radioprotective properties of cysteamine may mitigate a stress reaction of the pituitary-adrenal system occurring shortly after irradiation 8 references.
IT 60-23-1
RL: BIOL (Biological study)
(in radiation-damage prevention, adrenal glands in relation to)
RN 60-23-1 CAPLUS
CN Ethanethiol, 2-amino- (8CI, 9CI) (CA INDEX NAME)

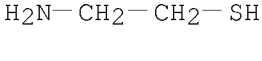


L4 ANSWER 71 OF 88 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1958:51042 CAPLUS
DOCUMENT NUMBER: 52:51042
ORIGINAL REFERENCE NO.: 52:9252d-f
TITLE: Action of drugs on the adrenal response of the rat to total-body x-irradiation
AUTHOR(S): Bacq, Z. M.; Fischer, P.
CORPORATE SOURCE: Univ. Liege, Belg.
SOURCE: Radiation Research (1957), 7, 365-72
CODEN: RAREAE; ISSN: 0033-7587
DOCUMENT TYPE: Journal
LANGUAGE: Unavailable
AB cf. C.A. 49, 11756a, 13500i. Drugs such as morphine and barbiturates inhibited the first adrenal reaction of rats to lethal doses of x-rays, but did not affect the second reaction or decrease mortality. Prior to irradiation, intraperitoneal injections of nembutal followed by morphine prevented a change in ascorbic acid and cholesterol values in the adrenals. It was suggested that the first adrenal response is a simple reversible reaction to phys. stress from irradiation. The second irreversible reaction, which was inhibited by cysteamine, seems to be related to the general deterioration of the irradiated rats.
IT 60-23-1, Ethanethiol, 2-amino-
(in x-ray damage prevention, to adrenal glands)
RN 60-23-1 CAPLUS

CN Ethanethiol, 2-amino- (8CI, 9CI) (CA INDEX NAME)



L4 ANSWER 72 OF 88 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1957:44385 CAPLUS
DOCUMENT NUMBER: 51:44385
ORIGINAL REFERENCE NO.: 51:8293a-c
TITLE: Antithyroid action and molecular structure. Sulfurated amino acids and derivatives
AUTHOR(S): Cheymol, Jean; Delsol, Michel; Durey, J. M.
CORPORATE SOURCE: Hopital Tenon, Paris
SOURCE: Annales Pharmaceutiques Francaises (1956), 14, 635-9
CODEN: APFRAD; ISSN: 0003-4509
DOCUMENT TYPE: Journal
LANGUAGE: Unavailable
AB The effect on thyroid and adrenals of injections of cysteine-HCl, cysteinamine-HCl, homocysteine, and methionine in equimol. quantities has been studied in rats. Daily treatment was given for 15 days. Cysteine and cysteinamine did not modify the thyroids. The weight of the adrenals was increased but this can be ascribed to a stress action because the injections caused local tissue damage. Homocysteine caused a moderate decrease in thyroid weight This can be due to an inhibition of the formation of thyrotropic hormone in the pituitary, but is not a goitrogenic effect. Methionine had no effect on the thyroid and apparently a slight stress effect on the adrenals.
IT 60-23-1, Ethanethiol, 2-amino-
(effect on adrenal and thyroid glands)
RN 60-23-1 CAPLUS
CN Ethanethiol, 2-amino- (8CI, 9CI) (CA INDEX NAME)



L4 ANSWER 73 OF 88 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2003:396264 CAPLUS
DOCUMENT NUMBER: 138:406594
TITLE: Topical skin composition for prevention of adverse or detrimental effects of ROS
INVENTOR(S): Mayne, James R.
PATENT ASSIGNEE(S): Access Business Group International LLC, USA
SOURCE: U.S. Pat. Appl. Publ., 12 pp., Cont.-in-part of Appl. No. PCT/US00/31933.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 4
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 20030095959	A1	20030522	US 2002-155305	20020524 <--
WO 2001037788	A1	20010531	WO 2000-US31933	20001121 <--

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR,

HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT,
LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU,
SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN,
YU, ZA, ZW
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY,
DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF,
BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

US 20070003536	A1	20070104	US 2006-497152	20060731
US 20080081082	A1	20080403	US 2006-617871	20061229
US 20080081034	A1	20080403	US 2006-617884	20061229
US 20080124409	A1	20080529	US 2006-617890	20061229

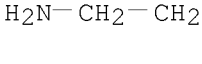
PRIORITY APPLN. INFO.:

WO 2000-US31933	A2 20001121
US 1999-167539P	A2 19991124
US 2002-155305	A2 20020524
US 2006-497152	A1 20060731

AB A topical skin composition that includes a complex containing an effective amount of selected components to provide a defense against the various pathway mechanisms of reactive oxygen species (ROS) is described. The complex composition contains components that counteract the reactive oxygen species reaction involving superoxide, hydrogen peroxide, and hydroxy reactions, and optionally at least one chain breaker component. In addition, a method for the treatment of the skin is provided.

IT 60-23-1, Cysteamine
RL: COS (Cosmetic use); BIOL (Biological study); USES (Uses)
(topical skin composition for prevention of adverse or detrimental effects of reactive oxygen species)

RN 60-23-1 CAPLUS
CN Ethanethiol, 2-amino- (8CI, 9CI) (CA INDEX NAME)



L4 ANSWER 74 OF 88 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2000:30039 CAPLUS
DOCUMENT NUMBER: 132:162201
TITLE: Cysteamine pretreatment of the astroglial substratum (mitochondrial iron sequestration) enhances PC 12 cell vulnerability to oxidative injury
AUTHOR(S): Frankel, Dov; Schipper, Hyman M.
CORPORATE SOURCE: Bloomfield Center for Research in Aging, Lady Davis Institute for Medical Research, Sir Mortimer B. Davis Jewish General Hospital, McGill University, Montreal, QC, H3T-1E2, Can.
SOURCE: Experimental Neurology (1999), 160(2), 376-385
CODEN: EXNEAC; ISSN: 0014-4886
PUBLISHER: Academic Press
DOCUMENT TYPE: Journal
LANGUAGE: English

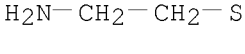
AB Much of the excess iron reported in the substantia nigra of subjects with Parkinson's disease (PD) implicates nonneuronal (glial) cellular compartments. Yet, the significance of these glial iron deposits vis-a-vis toxicity to indigent nigrostriatal dopaminergic neurons remains unclear. Cysteamine (CSH) induces the appearance of iron-rich (peroxidase-pos.) cytoplasmic inclusions in cultured rat astroglia, which are identical to glial inclusions that progressively accumulate in substantia nigra and other subcortical brain regions with advancing age. We previously demonstrated that the iron-mediated peroxidase activity in

these cells oxidizes dopamine and other catechols to potentially neurotoxic semiquinone radicals. In the present study, we cocultured catecholamine-secreting PC12 cells (as low-d. dispersed cells or high-d. colonies) atop monolayers of either CSH-pretreated (iron-enriched) or control rat astroglial substrata. In some expts., the PC12 cells were differentiated with nerve growth factor (NGF). The nature of the glial substratum did not appreciably affect the growth characteristics of the PC12 cells. However, undifferentiated PC12 cells grown atop CSH-pretreated astrocytes (a senescent glial phenotype) were far more susceptible to dopamine (1 μM)-H2O2(1 μM)-related killing than PC12 cells cultured on control astroglia. Differentiated PC12 cells behaved similarly although the fraction killed was about half that seen with the undifferentiated PC12 cells. In the latter expts., PC12 cell death was abrogated by coadministration of the antioxidants, ascorbate (200 μM), melatonin (100 μM), or resveratrol (50 μM) or the iron chelator, deferoxamine (400 μM), attesting to the role of oxidative stress and catalytic iron in the mechanism of PC12 cell death in this system. The aging-associated accumulation of redox-active iron in subcortical astrocytes may facilitate the bioactivation of dopamine to neuronotoxic free radical intermediates and thereby predispose the senescent nervous system to PD and other neurodegenerative disorders. (c) 1999 Academic Press.

IT 60-23-1, Cysteamine
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(cysteamine pretreatment of astroglial substratum (mitochondrial iron sequestration) enhances PC 12 cell vulnerability to oxidative injury)

RN 60-23-1 CAPLUS

CN Ethanethiol, 2-amino- (8CI, 9CI) (CA INDEX NAME)



REFERENCE COUNT: 50 THERE ARE 50 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 75 OF 88 CAPLUS COPYRIGHT 2008 ACS on STN

ACCESSION NUMBER: 2003:314737 CAPLUS

DOCUMENT NUMBER: 140:73499

TITLE: Design and analysis of microcantilevers for biosensing applications

AUTHOR(S): Zhang, Xuan; Yang, Mo; Ozkan, Cengiz S.

CORPORATE SOURCE: Mechanical Engineering Department, University of California, Riverside, CA, 92521, USA

SOURCE: Materials Research Society Symposium Proceedings (2003), 738(Spatially Resolved Characterization of Local Phenomena in Materials and Nanostructures), 375-380
CODEN: MRSPDH; ISSN: 0272-9172

PUBLISHER: Materials Research Society

DOCUMENT TYPE: Journal

LANGUAGE: English

AB The primary deflection due to the chemical reaction between the analyte mols. and the receptor coating, which produces surface stresses on the receptor side is analyzed. The resonance frequency of microcantilevers is very sensitive to the properties of the microcantilever surface. Biosensing expts. based on resonance frequency shift are presented, which show that the results strongly depend on the interaction of specific analyte mols. with the receptor surface.

IT 60-23-1

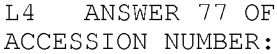
RL: ARU (Analytical role, unclassified); BUU (Biological use, unclassified); ANST (Analytical study); BIOL (Biological study); USES (Uses)
(self-assembled monolayer; design and anal. of microcantilevers for biosensing applications)
RN 60-23-1 CAPLUS
CN Ethanethiol, 2-amino- (8CI, 9CI) (CA INDEX NAME)

$H_2N—CH_2—CH_2—SH$

REFERENCE COUNT: 14 THERE ARE 14 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

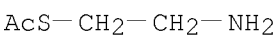
L4 ANSWER 76 OF 88 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2003:142294 CAPLUS
DOCUMENT NUMBER: 138:366873
TITLE: Methylglyoxal metabolism and diabetic complications: roles of aldose reductase, glyoxalase-I, betaine aldehyde dehydrogenase and 2-oxoaldehyde dehydrogenase
AUTHOR(S): Vander Jagt, David L.; Hunsaker, Lucy A.
CORPORATE SOURCE: Department of Biochemistry and Molecular Biology, University of New Mexico School of Medicine, Albuquerque, NM, 87131, USA
SOURCE: Chemico-Biological Interactions (2003), 143-144, 341-351
CODEN: CBINA8; ISSN: 0009-2797
PUBLISHER: Elsevier Science Ireland Ltd.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The 2-oxoaldehyde methylglyoxal (MeG) is the precursor to a number of the known advanced glycation end-products (AGE) implicated in the development of diabetic complications. Other 2-oxoaldehydes that are important in AGE formation, such as glyoxal, glucosone, deoxyglucosone, xylosone and deoxyxylosone, are produced by nonenzymic reactions. By contrast, MeG is produced by both enzymic and nonenzymic processes, most of which appear to be enhanced in diabetes. MeG may be a major precursor to formation of AGE, and rates of production of MeG depend upon physiol. conditions such as hyperglycemia and ketoacidosis. MeG is also unique compared to the other 2-oxoaldehydes in its complex metabolism At least four pathways contribute to detoxification of MeG:. (1) Aldose reductase, a member of the aldo-keto reductase superfamily, catalyzes the NADPH-dependent reduction of a wide range of aldehydes. MeG is the best of the known physiol. aldehyde substrates of aldose reductase. The distribution of aldose reductase in human tissue is restricted; there is little expression in liver. (2) The ubiquitous and highly active glyoxalase system converts MeG into d-lactate. However, this system depends upon the availability of glutathione; activity is severely limited by conditions of oxidative stress that impact levels of glutathione. (3) Betaine aldehyde dehydrogenase, also known as ALDH9, is able to catalyze the oxidation of MeG to pyruvate, although less efficiently than with its substrate betaine aldehyde. (4) The long-known but not widely studied 2-oxoaldehyde dehydrogenases (2-ODHs) catalyze the oxidation of MeG to pyruvate, primarily in liver. There are two NADP-dependent 2-ODHs in human liver. Both of these require an activating amine. The physiol. activator is unknown.
IT 60-23-1, 2-Aminoethanethiol
RL: BSU (Biological study, unclassified); BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)
(purification of 2 forms of 2-oxoaldehyde dehydrogenases from human liver, its substrate specificity, and amine dependence in relation to

methylglyoxal and diabetic complications)
RN 60-23-1 CAPLUS
CN Ethanethiol, 2-amino- (8CI, 9CI) (CA INDEX NAME)



REFERENCE COUNT: 56 THERE ARE 56 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

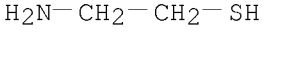
L4 ANSWER 77 OF 88 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2001:321166 CAPLUS
DOCUMENT NUMBER: 135:190367
TITLE: NAC/MEA Conjugate: A New Potent Antioxidant which Increases the GSH Level in Various Cell Lines
AUTHOR(S): Oiry, J.; Mialocq, P.; Puy, J. Y.; Fretier, P.; Clayette, P.; Dormont, D.; Imbach, J. L.
CORPORATE SOURCE: Sciences et Techniques du Languedoc, Laboratoire de Chimie Organique Biomoleculaire de Synthese, UMR 5625 CNRS-UM II, Universite Montpellier II, Montpellier, 34095, Fr.
SOURCE: Bioorganic & Medicinal Chemistry Letters (2001), 11(9), 1189-1191
CODEN: BMCLE8; ISSN: 0960-894X
PUBLISHER: Elsevier Science Ltd.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB I-152 is a prodrug of NAC and MEA with potent pro-GSH effects in human macrophages, astrocytes and lymphocytes. This mol. could be of interest in HIV infection in respect to its antioxidant and anti-HIV activities, but also in other diseases to counteract oxidative stress, i.e., inflammation, cardiovascular diseases, and neurodegenerative diseases. The NAC/MEA conjugate I-152 is a potent non-toxic antioxidant which increases the GSH level in various cell lines. This compound also presents an anti-HIV effect in the micromolar range.
IT 6197-31-5, S-Acetylcysteamine
RL: RCT (Reactant); RACT (Reactant or reagent)
(antioxidant acetylcysteine-cysteamine conjugate: effect on glutathione levels)
RN 6197-31-5 CAPLUS
CN Ethanethioic acid, S-(2-aminoethyl) ester (CA INDEX NAME)



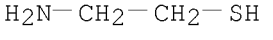
REFERENCE COUNT: 30 THERE ARE 30 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 78 OF 88 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1999:371651 CAPLUS
DOCUMENT NUMBER: 131:127476
TITLE: Induction proteins by cysteamine in Escherichia coli cells
AUTHOR(S): Suslov, A. V.; Suslova, I. N.
CORPORATE SOURCE: B.P. Konstantinov St.-Petersburg Institute of Nuclear Physics, Academy of Sciences of Russia, Gatchina, Russia
SOURCE: Radiatsionnaya Biologiya, Radioekologiya (1998), 38(4), 488-494

CODEN: RBIREJ; ISSN: 0869-8031
PUBLISHER: Nauka
DOCUMENT TYPE: Journal
LANGUAGE: Russian
AB Cysteamine, in presence of oxygen in broad interval time of treatment in E. coli cells, induced a series of proteins of SOS-repair system and heat-shock system. There are quant. defined induction levels of RecA, GroEL and DnaK proteins which are members or these systems. It was proposed that radioprotective action of cysteamine is defined by its characteristics as induction agent for stress systems of cells.
IT 60-23-1
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(induction proteins by cysteamine in Escherichia coli cells)
RN 60-23-1 CAPLUS
CN Ethanethiol, 2-amino- (8CI, 9CI) (CA INDEX NAME)



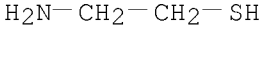
L4 ANSWER 79 OF 88 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1995:628194 CAPLUS
DOCUMENT NUMBER: 123:108820
ORIGINAL REFERENCE NO.: 123:19339a,19342a
TITLE: Differential effects of cysteamine on heat shock protein induction and cytoplasmic granulation in astrocytes and glioma cells
AUTHOR(S): Chopra, Vikramjit S.; Chalifour, Lorraine E.; Schipper, Hyman M.
CORPORATE SOURCE: Bloomfield Centre for Research in Aging, Lady Davis Institute for Medical Research, Sir Mortimer B. Davis, Jewish General Hospital, 3755 chemin Cote Ste. Catherine, Montreal, Que. H3T 1E2, Can.
SOURCE: Molecular Brain Research (1995), 31(1,2), 173-84
CODEN: MBREE4; ISSN: 0169-328X
PUBLISHER: Elsevier
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The sulfhydryl agent, cysteamine (CSH), promotes the accumulation of autofluorescent, peroxidase-pos. cytoplasmic granules in cultured astroglia akin to those which naturally accumulate in astrocytes of the aging periventricular brain. Both in vitro and in situ, CSH rapidly induces various heat shock proteins (HSP) in astrocytes long before granulation occurs. In the present study, we determined that CSH treatment resulted in an increase in HSP 27, HSP 90 and heme oxygenase (HO-1) at both the protein and mRNA level. We also showed that C6 glioma cells, unlike primary astrocytes, constitutively express HSP 27, HSP 90 and HO-1 at low levels. Moreover, CSH is incapable of eliciting further HSP expression or inducing granulation in the glioma cells. Our results support the hypothesis that the biogenesis of redox-active astrocytic inclusions in CSH-treated glial cultures and in the aging periventricular brain is dependent on an antecedent cellular stress response.
IT 60-23-1, Cysteamine
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(differential effects of cysteamine on heat shock protein induction and cytoplasmic granulation in astrocytes and glioma cells)
RN 60-23-1 CAPLUS
CN Ethanethiol, 2-amino- (8CI, 9CI) (CA INDEX NAME)



L4 ANSWER 80 OF 88 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1989:490195 CAPLUS
DOCUMENT NUMBER: 111:90195
ORIGINAL REFERENCE NO.: 111:14992h,14993a
TITLE: Cytoprotective and antiulcer activities of the antacid magaldrate in the rat
AUTHOR(S): Borella, L. E.; DiJoseph, J. F.; Mir, G. Nabi
CORPORATE SOURCE: Wyeth-Ayerst Res., Princeton, NJ, 08540, USA
SOURCE: Arzneimittel-Forschung (1989), 39(7), 786-9
CODEN: ARZNAD; ISSN: 0004-4172
DOCUMENT TYPE: Journal
LANGUAGE: English

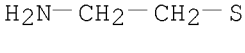
AB The cytoprotective and antiulcer activities of the antacid magaldrate (ES Riopan) as well as its effects on gastric mucosal blood flow and mucus secretions, were determined in the rat. Magaldrate afforded protection against gastric necrotic lesions induced by absolute ethanol (ED50, as magaldrate, 419 mg/kg); gastric ulcers induced by acidified acetylsalicylic acid (ED50 540 mg/kg), stress (cold restraint, ED50 388 mg/kg), indomethacin (ED50 281 mg/kg), and pylorus ligation; and intestinal ulcers induced by cysteamine (ED50 243 mg/kg) and indomethacin (ED50 184 mg/kg). At a dose of 8 mL/kg (1728 mg/kg magaldrate), the cytoprotective effect of magaldrate against ethanol was evident 1 min after oral administration and lasted more than 8 h. The cytoprotection induced by magaldrate was decreased by pretreatment with the depletor of endogenous thiols, n-ethylmaleimide, or with the cyclooxygenase inhibitor, indomethacin. Magaldrate did not affect gastric mucosal blood flow, but it increased gastric mucous secretion. This later effect may be a factor responsible for the cytoprotective activity of the agent. The efficacy of magaldrate may be due not only to its antacid, bile sequestering, and antipeptic activities, but also to its cytoprotective activity. The present results suggest that magaldrate could be effective in preventing gastric damage caused by alc. and antiinflammatory drugs.

IT 60-23-1, Cysteamine
RL: BIOL (Biological study)
(intestine damage from, magaldrate prevention of)
RN 60-23-1 CAPLUS
CN Ethanethiol, 2-amino- (8CI, 9CI) (CA INDEX NAME)



L4 ANSWER 81 OF 88 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1985:258 CAPLUS
DOCUMENT NUMBER: 102:258
ORIGINAL REFERENCE NO.: 102:47a,50a
TITLE: Chemosensitization: do thiols matter?
AUTHOR(S): Roizin-Towle, Laurie; Hall, Eric J.; Costello, Teresa; Biaglow, John E.; Varnes, Marie E.
CORPORATE SOURCE: Coll. Physicians Surg., Columbia Univ., New York, NY, 10032, USA
SOURCE: International Journal of Radiation Oncology, Biology, Physics (1984), 10(9), 1599-602
CODEN: IOBPD3; ISSN: 0360-3016

DOCUMENT TYPE: Journal
LANGUAGE: English
AB Thiol depletion as a mechanism responsible for enhanced cytotoxicity of melphalan [148-82-3] was assayed by pretreatment of cells in vitro with misonidazole [13551-87-6] and buthionine sulfoximine (BSO) [5072-26-4]. Hypoxic cell sensitizers, such as MISO, deplete endogenous thiols by metabolic activation under hypoxic conditions to thiol reactive intermediates, whereas BSO specifically inhibits a key enzyme in the synthesis of glutathione [70-18-8]. For a given level of thiol reduction, sensitization to melphalan was far greater by preincubation with MISO than it was for BSO. This indicated that thiol reduction itself was not the sole factor involved in chemosensitization by MISO. As evidence that the method of thiol depletion predisposes to the expression of biol. damage, it was shown that cells preincubated with MISO were appreciably more vulnerable to oxidative stress than those exposed to BSO. BSO was shown to totally inhibit the repair of damage from a preincubation treatment with MISO, demonstrating that recovery is dependent upon thiol regeneration. Thiol depletion "per se" is a good qual. but not necessarily a quant. indicator of chemosensitization, the biol. and biochem. function of the thiol depleting agents used influences further drug interactions. Thiols may play a potentially more critical role in the repair rather than the initiation of drug-induced damage.
IT 60-23-1
RL: BIOL (Biological study)
(chemosensitization to cytotoxic agents and thiol depletion in relation to)
RN 60-23-1 CAPLUS
CN Ethanethiol, 2-amino- (8CI, 9CI) (CA INDEX NAME)



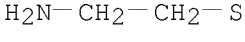
L4 ANSWER 82 OF 88 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1968:424963 CAPLUS
DOCUMENT NUMBER: 69:24963
ORIGINAL REFERENCE NO.: 69:4646h,4647a
TITLE: Neurosecretion in the hypothalamus and posterior pituitary after irradiation and injection of chemical radioprotectors in the rat
AUTHOR(S): Duchesne, P. Y.; Hajdukovic, S.; Beaumariage, M. L.; Bacq, Z. M.
CORPORATE SOURCE: Univ. Liege, Liege, Belg.
SOURCE: Radiation Research (1968), 34(3), 583-95
CODEN: RAREAE; ISSN: 0033-7587
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Acute whole-body exposure to 800-200 r. of 200-kv. x-rays induces instant activation of the neurosecretory processes in the hypothalamus and posterior hypophysis in rats, as observed by histochem. techniques. The same neurosecretory response is observed after exposures to 700 or 400 r. or x-rays delivered at an exposure rate of 10 r./min. as well as after exposures to 1000 or 800 r. of 137Cs γ-rays at 1 r./min. Partial irradiation of the head alone or irradiation of the animal body, with the head shielded, induces the same neurosecretory response. These results, added to previous information, suggest that the neurosecretory response to lethal or sublethal doses constitutes an important and necessary link in the stress reaction of mammals to ionizing radiation.
Cysteamine (150 mg./kg. of body weight in i.p. injection) and cystamine (same radioprotective dose) stimulate the neurosecretion of normal nonirradiated

rats. Subsequent exposure to x-rays (700 r. or 400 r.) further intensifies this process.

IT 60-23-1
RL: BIOL (Biological study)
(hypothalamus secretion in response to, after x-irradiation)

RN 60-23-1 CAPLUS

CN Ethanethiol, 2-amino- (8CI, 9CI) (CA INDEX NAME)



L4 ANSWER 83 OF 88 CAPLUS COPYRIGHT 2008 ACS on STN

ACCESSION NUMBER: 1967:418376 CAPLUS

DOCUMENT NUMBER: 67:18376

ORIGINAL REFERENCE NO.: 67:3475a,3478a

TITLE: Action of chemical radioprotective compounds on hypothalamic neurosecretion in rats exposed to ionizing radiation

AUTHOR(S): Duchesne, P. Y.; Hajdukovic, Srdjan

CORPORATE SOURCE: Univ. Liege, Liege, Belg.

SOURCE: Comptes Rendus des Seances de la Societe de Biologie et de Ses Filiales (1966), 160(11), 2207-8
CODEN: CRSBAW; ISSN: 0037-9026

DOCUMENT TYPE: Journal

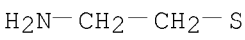
LANGUAGE: French

AB Administration of either β-mercaptoethylamine, cysteamine, or cystamine (150 mg./kg.) to rats caused an increase in hypothalamic neurosecretion. The administration of any of the 3 radioprotective compds. 10 min. prior to ionizing radiation (700 r. administered at 100 r./min.) caused the rats to react immediately to the radiation stress by an active secretion by the hypothalamic neurosecretory nuclei, a reaction which was found in untreated, irradiated rats only several hrs. after the irradiation

IT 60-23-1
RL: BIOL (Biological study)
(hypothalamus neurosecretion after irradiation and)

RN 60-23-1 CAPLUS

CN Ethanethiol, 2-amino- (8CI, 9CI) (CA INDEX NAME)



L4 ANSWER 84 OF 88 CAPLUS COPYRIGHT 2008 ACS on STN

ACCESSION NUMBER: 2007:1057559 CAPLUS

DOCUMENT NUMBER: 147:378363

TITLE: Methods for treating inflammatory disease by administering aldehydes and derivatives thereof

INVENTOR(S): De Matos, Marta N.; Romao, Carlos C.

PATENT ASSIGNEE(S): Alfama - Investigacao e Desenvolvimento de Productos Farmaceuticos Lda, Port.

SOURCE: U.S. Pat. Appl. Publ., 46pp., Cont.-in-part of U.S. Ser. No. 453,319.
CODEN: USXXCO

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 6

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 20070219120	A1	20070920	US 2007-702970	20070206
US 20040067261	A1	20040408	US 2003-356738	20030203 <--
US 7011854	B2	20060314		
US 20060148900	A1	20060706	US 2005-288670	20051129
US 20060233890	A1	20061019	US 2006-453319	20060614
WO 2008069688	A2	20080612	WO 2007-PT9	20070206
WO 2008069688	A3	20080731		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, GT, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN, MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RS, RU, SC, SD, SE, SG, SK, SL, SM, SV, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW

RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AP, EA, EP, OA

PRIORITY APPLN. INFO.:

US 2002-353233P	P	20020204
US 2003-356738	A3	20030203
US 2005-288670	A2	20051129
US 2006-453319	A2	20060614
US 2006-873155P	P	20061206

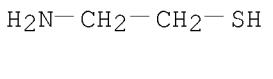
OTHER SOURCE(S): MARPAT 147:378363

AB A method is disclosed for treating inflammatory disease in an animal in need thereof by administering to the animal a pharmaceutical composition containing an anti-inflammatory effective amount of an organic aldehyde compound or a derivative thereof in a pharmaceutically acceptable vehicle. Aldehyde derivative prodrugs were prepared and administered to rat models of arthritis significantly reduced paw edema and improved the arthritic indexes in these animals. The tertiary aldehydes acted as carbon monoxide releasing mols. (CORMs) after exposure to reactive oxygen species.

IT 156-57-0, Cysteamine hydrochloride
RL: RCT (Reactant); RACT (Reactant or reagent)
(tertiary aldehyde derivs. for treating inflammatory disease)

RN 156-57-0 CAPLUS

CN Ethanethiol, 2-amino-, hydrochloride (1:1) (CA INDEX NAME)



L4 ANSWER 85 OF 88 CAPLUS COPYRIGHT 2008 ACS on STN

ACCESSION NUMBER: 2004:85774 CAPLUS

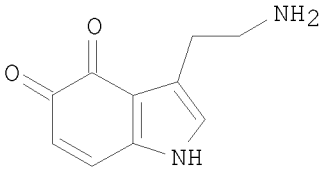
DOCUMENT NUMBER: 140:298813

TITLE: Reactions of the Putative Neurotoxin Tryptamine-4,5-dione with L-Cysteine and Other Thiols

AUTHOR(S): Jiang, Xiang-Rong; Wrona, Monika Z.; Alguindigue, Susan S.; Dryhurst, Glenn

CORPORATE SOURCE: Department of Chemistry and Biochemistry, University

of Oklahoma, Norman, OK, 73019, USA
SOURCE: Chemical Research in Toxicology (2004), 17(3), 357-369
CODEN: CRTOEC; ISSN: 0893-228X
PUBLISHER: American Chemical Society
DOCUMENT TYPE: Journal
LANGUAGE: English
GI



I

AB Tryptamine-4,5-dione (I) is formed by oxidation of 5-hydroxytryptamine by reactive oxygen and reactive nitrogen species. I is a powerful electrophile that can covalently modify cysteinyl residues of proteins and deactivate key enzymes. Thus, I has been suggested to play a role in the degeneration of serotonergic neurons in brain disorders such as Alzheimer's disease or evoked by amphetamine drugs. However, if formed in the brain, it is also likely that I would react with low mol. weight thiols such as cysteine (CySH) and glutathione (GSH). The resulting metabolites might not only contribute to the degeneration of serotonergic neurons but also, perhaps, serve as biomarkers of such neurodegeneration. In this investigation, it is shown that in oxygenated buffer at pH 7.4 I reacts with CySH and other low mol. weight sulfhydryls such as GSH, N-acetylcysteine, and cysteamine to form, first, the corresponding 7-S-thioethers of the dione. However, unlike the glutathionyl and N-acetylcysteinyl conjugates of I, the 7-S-cysteinyl conjugate is very unstable at pH 7.4 forming a number of novel products, the nature of which are dependent on the relative concns. of I and CySH. These products have been isolated, and spectroscopic and other evidence is provided in support of their proposed chemical structures.
IT 60-23-1, Cysteamine
RL: RCT (Reactant); RACT (Reactant or reagent)
(reactions of the putative neurotoxin tryptamine-4,5-dione with L-cysteine and other thiols)
RN 60-23-1 CAPLUS
CN Ethanethiol, 2-amino- (8CI, 9CI) (CA INDEX NAME)

$H_2N-CH_2-CH_2-SH$

REFERENCE COUNT: 65 THERE ARE 65 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 86 OF 88 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2001:128003 CAPLUS
DOCUMENT NUMBER: 135:41932
TITLE: Kinetics of the reactions of hypochlorous acid and amino acid chloramines with thiols, methionine, and ascorbate
AUTHOR(S): Peskin, A. V.; Winterbourn, C. C.

CORPORATE SOURCE: Department of Pathology, Free Radical Research Group, Christchurch School of Medicine, Christchurch, N. Z.
SOURCE: Free Radical Biology & Medicine (2001), 30(5), 572-579
CODEN: FRBMEH; ISSN: 0891-5849
PUBLISHER: Elsevier Science Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Thiol oxidation by hypochlorous acid and chloramines is a favorable reaction and may be responsible for alterations in regulatory or signaling pathways in cells exposed to neutrophil oxidants. In order to establish the mechanism for such changes, it is necessary to appreciate whether these oxidants are selective for different thiols as compared with other scavengers. We have measured rate consts. for reactions of amino acid chloramines with a range of thiols, methionine, and ascorbate, using a combination of stopped-flow and competitive kinetics. For HOCl, rate consts. are too fast to measure directly by our system and values relative to reduced glutathione were determined by competition with methionine. For taurine chloramine, the rate consts. for reaction with 5-thio-2-nitrobenzoic acid, GSH, methionine, and ascorbate at pH 7.4 were 970, 115, 39, and 13 M-1 s-1, resp. Values for 10 thiols varied by a factor of 20 and showed an inverse relationship to the pKa of the thiol group. Rate consts. for chloramines of glycine and N-α-acetyl-lysine also showed these relationships. Rates increased with decreasing pH, suggesting a mechanism involving acid catalysis. For hypochlorous acid, rates of reaction with 5-thio-2-nitrobenzoic acid, GSH, cysteine, and most of the other thiols were very similar. Relative reactivities varied by less than 5 and there was no dependence on thiol pKa. Chloramines have the potential to be selective for different cellular thiols depending on their pKa. For HOCl to be selective, other factors must be important, or its reactions could be secondary to chloramine formation.

IT 60-23-1, Cysteamine
RL: BSU (Biological study, unclassified); RCT (Reactant); BIOL (Biological study); RACT (Reactant or reagent)
(kinetics of reactions of hypochlorous acid and amino acid chloramines with thiols, methionine, and ascorbate)
RN 60-23-1 CAPLUS
CN Ethanethiol, 2-amino- (8CI, 9CI) (CA INDEX NAME)

$H_2N-CH_2-CH_2-SH$

REFERENCE COUNT: 43 THERE ARE 43 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 87 OF 88 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1997:104165 CAPLUS
DOCUMENT NUMBER: 126:182756
ORIGINAL REFERENCE NO.: 126:35205a,35208a
TITLE: Free radical reactions involving the angiotensin converting enzyme inhibitor captopril
AUTHOR(S): Forni, L. G.; Hilton, P. J.; Willson, R. L.; Cheeseman, K. H.
CORPORATE SOURCE: Department of Renal and Intensive Care Medicine, St Thomas' Hospital, London, UK
SOURCE: Redox Report (1996), 2(6), 393-399
CODEN: RDRPE4; ISSN: 1351-0002
PUBLISHER: Churchill Livingstone
DOCUMENT TYPE: Journal

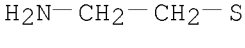
LANGUAGE: English

AB Using the pulse radiolysis technique, absolute rate consts. have been obtained for the reaction of captopril with several free radicals. The results demonstrate that although captopril reacts rapidly with a number of free radicals, such as the hydroxyl radical (K = 5.1 + 109 dm-3mol-1s-1) and the thiocyanate radical anion (k = 1.3 + 107 dm-3mol-1s-1), it is not exceptional in this ability. Similarly, the reactions with carbon centered radicals although rapid are an order of magnitude slower than those observed with glutathione. Addnl. lipid peroxidn. studies further demonstrate that captopril is a much less effective antioxidant than glutathione. The data go some way to supporting the view that any attenuation of reperfusion injury by captopril is not through a direct free radical scavenging mechanism but may be afforded by other, non-radical-mediated mechanisms.

IT 60-23-1, Cysteamine
RL: RCT (Reactant); RACT (Reactant or reagent)
(free radical reactions of the angiotensin converting enzyme inhibitor captopril and its antioxidant activity in in vitro lipid peroxidn. systems)

RN 60-23-1 CAPLUS

CN Ethanethiol, 2-amino- (8CI, 9CI) (CA INDEX NAME)



L4 ANSWER 88 OF 88 CAPLUS COPYRIGHT 2008 ACS on STN

ACCESSION NUMBER: 1995:917745 CAPLUS

DOCUMENT NUMBER: 124:81378

ORIGINAL REFERENCE NO.: 124:15145a,15148a

TITLE: Reversible introduction of thiol compounds into proteins by use of activated mixed disulfides

AUTHOR(S): Faulstich, Heinz; Heintz, Daniela

CORPORATE SOURCE: Max-Planck Institut fur Medizinische Forschung, Heidelberg, D-69120, Germany

SOURCE: Methods in Enzymology (1995), 251(Biothiols, Part A), 357-66
CODEN: MENZAU; ISSN: 0076-6879

PUBLISHER: Academic

DOCUMENT TYPE: Journal

LANGUAGE: English

AB The title topic is discussed with information on mixed disulfides and activated mixed disulfides (AMDs), AMDs of low mol. weight, advantages of derivatization of protein thiols with AMDs, monitoring unfolding of actin with AMDs, crosslinking of protein thiols with bifunctional AMDs, preparative methods for low-mol.-weight AMDs used for S-alkylthiolation of proteins, etc.

IT 156-57-0, Cysteamine hydrochloride
RL: RCT (Reactant); RACT (Reactant or reagent)
(reversible introduction of thiol compds. into proteins by using activated mixed disulfides)

RN 156-57-0 CAPLUS

CN Ethanethiol, 2-amino-, hydrochloride (1:1) (CA INDEX NAME)